Impact of a Social Skills Intervention on the Structural Anatomy of the Social Brain of Autistic Adolescents

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IMPACT OF A SOCIAL SKILLS INTERVENTION ON THE STRUCTURAL ANATOMY OF THE SOCIAL BRAIN OF AUTISTIC ADOLESCENTS

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ABSTRACT
IMPACT OF A SOCIAL SKILLS INTERVENTION ON THE
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The prevalence of Autism Spectrum Disorder (ASD) continues to rise as researchers seek to examine the physiological links to social and communication challenges. The Social Brain, neuroanatomical structures which play a role in social cognition, is proposed to be linked to the social and communication challenges associated with ASD. An area of rapidly growing research is the evaluation of social skills interventions, which target social challenges present in Autistic individuals. Advances in technology, have allowed for these interventions to be examined in regards to physiological changes (e.g., electroencephalogram asymmetry and coherence) as outcome variables. Amongst these interventions, the Program for Education and Enrichment of Relational Skills® has been shown to be efficacious across several countries and different cultural backgrounds.

The present study investigated neurophysiological outcomes in adolescents who receive PEERS®, compared to waitlist control and NT control groups. Structural magnetic resonance imaging (MRI) scans were utilized to evaluate changes in volume, cortical thickness, and surface area of adolescents across a randomized controlled-trial of PEERS®. We hypothesized that 1) Social Brain structures between NT and ASD groups would be significantly different at pre-intervention, with the exception of the Amygdala; 2) structural changes from pre- to post-intervention would be found in the ASD group that received PEERS®; and 3) significant structural changes found across time points would predict changes across time in questionnaire measures of social and communication challenges.

Results indicated: a) partial support of Hypothesis 1, showing that Amygdala volumes did not differ across groups, but contrary to our hypothesis the remaining structures also did not show a significant difference at pre-test between groups; b) partial support of Hypothesis 2, indicating bilateral amygdala, left caudal anterior cingulate cortex, and left superior temporal gyrus volumes changed differentially between groups over the course of PEERS®; c) Hypothesis 3 was not supported. In conclusion, this study is the first to indicate differential neuroanatomical volumetric changes over the course of a social skill intervention for Autistic individuals that received the intervention, contrasted to those that did not receive the intervention.

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I. Introduction

The prevalence of Autism Spectrum Disorder (ASD) continues to rise, with an estimated 1 in 59 children having a diagnosis (Baio et al., 2018). ASD, which is characterized by restricted, repetitive behaviors and challenges in social communication (American Psychiatric Association, 2013), is a neurodevelopmental disorder characterized by neurological differences in Autistic individuals in contrast to their Typically Developing (TD) counterparts (Barak & Feng, 2016; Courchesne, 2002; Ha et al., 2015; Herrington, Maddox, Kerns, et al., 2017; Lange et al., 2015; Petinou & Minaidou, 2017; Schumann et al., 2004, 2010). Furthermore, challenges in social communication and interaction are often associated with lower quantity and quality of friendships and increased levels of bullying and peer rejection, factors which have been linked to increased risk of internalizing comorbidity (e.g. Anxiety, and Depression; Mazurek & Kanne, 2010). This paper will provide an overview of ASD and typical development in adolescence, focusing on neurophysiology, social skills challenges in ASD, and comorbid symptoms in ASD linked to these social challenges. Subsequently, the current literature on the neurophysiological correlates of social behaviors will be reviewed. The current investigation will examine whether the morphology of neural structures subserving social behaviors are affected by a social skills treatment for adolescents with ASD.

II. ASD in Adolescence

Social skills challenges are characteristic of ASD throughout development (American Psychiatric Association, 2013) and become more pronounced during

adolescence, as it is a period of development marked by formation of meaningful relationships with others. Furthermore, the increasing demands of social ability, and the subsequent consequences of possessing or being deficient of those skills, may impact the further social development and adaptive functioning of an individual into adulthood (Picci & Scherf, 2015). The challenges in social communication that adolescents with ASD must confront as a part of daily life has been shown to decrease the quantity and quality of their friendships (Mazurek & Kanne, 2010), and lead to increased social isolation and feelings of loneliness (Deckers et al., 2017; Locke et al., 2010). These feelings of loneliness and social isolation have been shown to be linked to anxiety and depression (Locke et al., 2010; White & Roberson-Nay, 2009), with these heightened feelings of loneliness, relative to TD individuals, reported particularly during the period of adolescence (Deckers et al., 2017).

Anxiety and depression are two of the most frequent comorbidities found in Autistic individuals. Previous studies have estimated rates of anxiety comorbidity ranging from 11% to 84% (Simonoff et al., 2008; van Steensel et al., 2011, 2013; White et al., 2009), with social phobia, obsessive compulsive disorder, and social anxiety being the most common anxiety comorbidities in ASD found in a prior meta-analysis (van Steensel et al., 2011). Symptoms of depression are, likewise, common, with variable estimates of prevalence. Prior studies have reported clinically elevated depression symptoms in 10.9% to 54% of Autistic individuals (J. A. Kim et al., 2000; Mayes, Calhoun, Murray, Ahuja, et al., 2011; Simonoff et al., 2008). Additional investigation of depression in ASD has shown increasing depressive symptoms to be associated with increasing age and IQ (Mayes, Calhoun, Murray, & Zahid, 2011). Furthermore, challenges in social functioning

and communication have been linked to greater symptoms of depression, including increased risk of suicidal ideation and self-harm (Culpin et al., 2018). A common posited explanation for these links between depression and IQ, and depression and social functioning, is that Autistic individuals without cognitive disability have a greater self-awareness of their challenges in social communication, but lack the resources to improve their social functioning, and therefore, as aforementioned, have fewer friendships (DeFilippis, 2018; Mazurek & Kanne, 2010). Thus, it may be that by targeting social difficulties in adolescents with ASD, depression and anxiety, particularly social anxiety, may be ameliorated by creating a protective factor against these comorbid symptoms. However, these internalizing symptoms may be difficult to assess from an overt behavioral standpoint, making it particularly challenging for a clinician, caregiver, or other third-party to objectively assess change in symptoms across time. Social skills and difficulties, although more visible than depression and anxiety, are also often assessed using questionnaires and interviews, which are subject to several biases (e.g., expectancy bias and investigator bias; Choi & Pak, 2004; Rutherford, Rose, Sneed, & Roose, 2009). However, previous research has explored the link between mental health disorders and neurophysiology. This line of research has provided what may a be step closer to objectively quantifying change across an intervention.

A. Social Brain

From electrical currents conducting the movement and function of inter-neuronal activity, to readily-observable behaviors such as speaking, the causal links from neurophysiology to overt behavior are undeniable, yet vastly complex, thus remaining a

subject of investigation. The concept of the "Social Brain," a set of brain regions dedicated to social cognition and behavior, was first put forth almost three decades ago (Brothers, 1990). The proposition stemmed from the effects observed on social behavior, resultant from brain lesions or single neuron studies on specific areas of primate brains (Brothers, 1990, 1996). At the time of origin, the "Social Brain" was proposed to be composed of the amygdala, orbitofrontal cortex, and temporal cortex, particularly the superior temporal sulcus, due to the aforementioned lesion studies. Since its inception, the "Social Brain" has been vastly researched (e.g., Blakemore, 2008; McPartland & Pelphrey, 2012; Pua, Bowden, & Seal, 2017; Rojas et al., 2006; Whyte, Behrmann, Minshew, Garcia, & Scherf, 2016), and the concept has evolved to be hypothesized to consist of several more structures (McPartland & Pelphrey, 2012). The fusiform gyrus (FFG) or fusiform face area (FFA), Insula, Anterior Cingulate Cortex (ACC), and the Medial Prefrontal Cortex (mPFC) are among the notable additions to the "Social Brain" (Adolphs, 2009; Chen et al., 2018; Frith, 2007; McPartland & Pelphrey, 2012; Uddin et al., 2017). Since the time Brothers (1990) first proposed the Social Brain, there has also been a large portion of research dedicated to comparing and contrasting the Autistic brain with the typically developing brain both in terms of structure/volume (Groen et al., 2010; Herrington, Maddox, Kerns, et al., 2017; Kohli et al., 2018; Lange et al., 2015; Petinou & Minaidou, 2017; Prigge et al., 2018; Rojas et al., 2006; Schumann et al., 2004, 2009, 2010) and "functional activation" (e.g., Ha et al., 2015; Herrington, Maddox, McVey, et al., 2017; Hileman, Henderson, Mundy, Newell, & Jaime, 2011; Parellada et al., 2014; Petinou & Minaidou, 2017; Van Hecke et al., 2015). Furthermore, investigations have been initiated to examine the differences in structure/volume and "functional activation"

across TD samples and samples with depression or anxiety, especially as regards the amygdala (e.g., Hamilton, Siemer, & Gotlib, 2008; Herrington, Maddox, Kerns, et al., 2017; Machado-de-Sousa et al., 2014; McEwen, 2003). This research is pivotal to understanding any brain differences in Autistic individuals, given the high comorbidity of depression and anxiety, and the effects those comorbid symptoms have on structures of the Social Brain, such as the amygdala.

i. Amygdala. The amygdala is a structure that has been implicated in contributing to symptoms of several clinical mental health disorders (Schumann et al., 2011); amongst them being clinical presentations of ASD, anxiety, and depression (Groen et al., 2010; Hamilton et al., 2008; Herrington, Maddox, Kerns, et al., 2017; Herrington, Maddox, McVey, et al., 2017; Machado-de-Sousa et al., 2014; Nacewicz et al., 2006; Schumann et al., 2004). An early study showed amygdala lesions in Rhesus monkeys resulted in social behavioral changes, with the lesioned monkeys showing decreased appropriate emotional responses (e.g., situation-appropriate aggression) and an overall indifference to social situations (Dicks et al., 1969). Later studies showed single neuron activity within the amygdala of Macaque monkeys when presented with facial expressions and social situations (Brothers et al., 1990; Leonard et al., 1985; David I. Perrett & Mistlin, 1990). Since these early studies, the links between social behaviors and psychiatric symptoms and the amygdala have been furthered explored with human studies commonly utilizing functional MRI (*f*MRI), and volumetric measures in their investigations (e.g., Herrington, Maddox, Kerns, et al., 2017; Nacewicz et al., 2006; Schumann et al., 2011, 2004). Although a few steps removed from single neuron recordings, structural volume is commonly used in neuroanatomy research as an analogue for absolute neuron count, thus

greater processing capabilities, and has been consistently shown to be positively associated with volume of a structure in primates (e.g., Herculano-Houzel, 2009, 2012; Im et al., 2008; Roth & Dicke, 2005). Therefore, using a variety of methods (e.g. *f*MRI, and structural volume), the amygdala has been posited to be involved in analyzing facial expressions to recognize the emotional states of others (e.g., Santos, Almeida, Oliveiros, & Castelo-Branco, 2016; Williams, Morris, McGlone, Abbott, & Mattingley, 2004), as well as helping to experience and regulate one's own emotion (e.g., Morawetz, Alexandrowicz, & Heekeren, 2017), therefore facilitating fluent and appropriate social-emotional interactions.

Within the scope of ASD, findings concerning amygdalae volumes in adolescence are mixed. Whilst in children with ASD, compared to their TD counterparts, there are mostly consistent findings of increased volume (Bellani et al., 2013; J. E. Kim et al., 2010; Mosconi et al., 2009; Nordahl et al., 2012; Schumann et al., 2004), results are mixed in studies in adolescence. Whereas some studies show a continued significantly enlarged amygdala in Autistic adolescents in contrast to their TD peers (Groen et al., 2010; Howard et al., 2000; Mosconi et al., 2009; Munson et al., 2006), others show significantly decreased volumes (Aylward et al., 1999; Nacewicz et al., 2006; Pierce et al., 2001; van Rooij et al., 2018) or no difference between the groups (Haznedar et al., 2000; Nacewicz et al., 2006; Schumann et al., 2004).

There are a few studies which have examined the relational link between amygdala volume at different ages and various social behaviors. However, the relation between amygdala volume and social skills deficits in adolescence is relatively sparse in contrast to other age groups, as well as compared to other examinations on amygdala

volume in relation with psychiatric symptoms (e.g., anxiety and depression; Hamilton et al., 2008; Machado-de-Sousa et al., 2014; Morey et al., 2012).

Specifically, amongst the extant literature of amygdala volume links with social behavior, most studies have focused on children or adults (e.g., Baribeau et al., 2019; Bickart, Wright, Dautoff, Dickerson, & Barrett, 2011; Corbett et al., 2009; Dziobek, Fleck, Rogers, Wolf, & Convit, 2006; Juranek et al., 2006; Mitchell et al., 2009; Mosconi et al., 2009; Munson et al., 2006; Schumann et al., 2009; van Rooij et al., 2018), whilst few (Baribeau et al., 2019; Nacewicz et al., 2006) have delved into examining adolescence. Amongst the first to report in this area was Munson and colleagues (2006), in an investigation of three to four year-olds with ASD. Larger right amygdala volumes were found to be not only associated with greater difficulties on social and communication impairments, measured using the Vineland social and communication domains, a well-validated assessment for developmental and intellectual disabilities in which lower scores indicate greater challenges (Perry & Factor, 1989), but also predictive of social deficits at age six, above and beyond IQ and total brain volume. A different study that same year, utilizing children with ASD, found no association between amygdala volumes and the communication and social subdomains of the Autism Diagnostic Observation Schedule (ADOS; Juranek et al., 2006; Lord et al., 2000). However, it is important to note that although the ADOS and the Vineland both contain communication and social domains and have overlap with some of their coding, the Vineland also assess several aspects the ADOS does not, such as “playing with a peer.” Therefore, it may be that the discrepancies in these two aforementioned studies are due to the differences between social and communicative behaviors captured by the ADOS

versus the Vineland. Nonetheless, a later study published results showing a positive association between ADOS-G scores and amygdala size, thus greater difficulties (i.e., higher ADOS-G scores) associated with larger amygdala size (Mitchell et al., 2009). It should be noted that Mitchell and colleagues had a more diverse age sample, with some participants falling in the adolescent age range, but did not control for age in their analysis. Hence, the contradictory results of these studies (i.e., Juranek et al., 2006; Mitchell et al., 2009) may have been due to age variability in their samples. Further complication of a consistency in findings on amygdala volumes and ADOS scores is shown in a meta-analysis reporting a negative association between the two factors (van Rooij et al., 2018); thus contradicting the findings by Mitchell et al on amygdala relations with the ADOS and Munson et al., on amygdala relations with Vineland social and communication scores.

Further, the Munson et al. (2006) findings were later replicated, as well as extended, to show a positive relation between amygdala size and Autism Diagnostic Interview Revised (ADI-R) social and nonverbal (i.e., non-vocal verbal) communication (Schumann et al., 2009), thus larger amygdala volumes were associated with greater challenges (i.e., higher ADI-R scores). Schumann and colleagues (2009) also showed that male participants were driving the significant associations and that females did not show any significant associations with either the Vineland or the ADI-R. Mosconi and colleagues (2009) examined the relation of amygdala volumes and joint attention in toddlers, a skill deficit often found in Autistic children (American Psychiatric Association, 2013; Mundy & Newell, 2007). Joint attention must be acquired prior to skill acquisition of certain higher-order social behaviors, such as appropriately initiating a

jointly-focused conversation. Hence, there is importance in understanding the neurobiological underpinnings of possessing or having challenges with joint attention. Results from this study provided evidence for a positive association between amygdala volumes in Autistic two to four year old children and joint attention (i.e., larger volumes were linked to greater joint attention; Mosconi et al., 2009).

Although the findings of a relation between larger amygdala size and greater social and communicative difficulties are mostly consistent, the findings in adolescent samples are contradictory to the findings in children. A recent study examining subcortical structure volumes and relations with the Social Communication Questionnaire (SCQ; a common questionnaire completed by parents and used to quantify ASD symptoms in their child; Berument, Rutter, Lord, Pickles, & Bailey, 1999; Rutter, Bailey, & Lord, 2003) and the Reading the Mind in the Eyes Test (RMET; a test which ask individuals to evaluate the emotion presented in an image of a single eye; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001; Baron-Cohen, Wheelwright, Spong, Scahill, & Lawson, 2001) across diagnostic groups and TD young adolescents found the following: (a) There were no amygdala volume differences across their ASD and TD sample; (b) Smaller left amygdala volumes predicted greater difficulties in social communication measured by the SCQ; and (c) Smaller amygdala sizes were predictive of greater challenges in emotion recognition measured by the RMET (Baribeau et al., 2019). An investigation from Nacewicz and colleagues (2006), which examined adolescents and young adults in two separate studies, found similar results to Baribeau and colleagues (2019). Both studies revealed: (a) Smaller amygdala volume predicted lower eye fixation fraction (i.e., eye fixation time divided by total face fixation time) in the Autistic samples,

but not in the TD samples; and (b) Smaller amygdala volumes were predictive of greater difficulties in social reciprocity and nonverbal (non-vocal) communication in both TD and Autistic samples. Furthermore, upon combining their samples from both studies, it was revealed that right amygdala volumes were driving the significant prediction of eye fixation fraction. However, perhaps the most important revelations from the combined samples analysis were two-fold. First, eye fixation time showed an interaction with age and amygdala volume with eye fixation increasing with age and amygdala volume. Second, differential patterns in the Autistic sample contrasted to the TD sample emerged when examining the aforementioned interaction. Whilst the TD sample showed a steady increase in age, amygdala volume and eye fixation volume, the ASD group showed that that younger participants showed a similar growth pattern until about age 14.8 years. At this age, two distinct patterns emerged in the ASD sample: (1) Individuals who showed eye fixation time similar to their TD counterparts showed the same age-related increase in amygdala volume as TD; and (2) Individuals with ASD who showed lower eye fixation also showed smaller increases in amygdala size with age increase (Nacewicz et al., 2006).

Adult literature is also limited in regards to the aforementioned relational investigations. However, a study utilizing a TD adult sample also found a positive association between amygdalae volumes and a person's respective social network, both in size of a person's social network (i.e., total number of people in social network with whom the participant had regular contact), as well as its complexity (i.e. total number of different groups to which the contacts in the social network belonged; Bickart et al., 2011). In males (n = 36), specifically, this link was significant for the complexity and left

amygdala volume, and nearing significance for predictive value of left amygdala volume and social network size. Therefore, the extant literature on amygdalae volume and their relation to social and communication skills, although for the most part consistent within age groups, show a discordant pattern across age groups. Specifically, while larger amygdala volumes in children are related to greater social difficulties, the inverse is found (i.e., larger amygdala volumes are related to less social difficulties) in adolescents and young adults.

ii. Superior Temporal Sulcus/Gyrus. A structure of additional relevance in the investigation of social communication and cognition is the temporal cortex, particularly the superior temporal region. Two regions, the superior temporal sulcus (STS) and superior temporal gyrus (STG), have been linked to the abovementioned social abilities (Nathalie Boddaert & Zilbovicius, 2002; Jou et al., 2010; McPartland & Pelphrey, 2012). Located between the superior and medial temporal gyri of the temporal cortex, the STS has been reported to play a vital role in analyzing biological visual motion stimuli (e.g., eye and hand movements) and to use this analysis to predict further movement, as well as intention (i.e., theory of mind), of people in the environment (e.g., Allison, Puce, & McCarthy, 2000; Bonda, Petrides, Ostry, & Evans, 1996; Pelphrey, Singerman, Allison, & McCarthy, 2003; D. I. Perrett et al., 1989; Puce & Perrett, 2003). Furthermore, through investigations of neural activation with the use of *f*MRIs, Event-Related Potentials (ERP), and positron emission tomography (PET) scans, it has been posited that the STS plays a crucial role in the mirror neuron system (MNS; e.g., Jeon & Lee, 2018; Molenberghs, Brander, Mattingley, & Cunnington, 2010; Pineda, 2005, 2008), a system of neurons activated by observing another's actions and duplicating observed actions that are

executed by the mouth, hands, and/or arms in non-human primates (e.g., Murata et al., 1997; Raos, Umiltá, Murata, Fogassi, & Gallese, 2006; Rozzi, Ferrari, Bonini, Rizzolatti, & Fogassi, 2008). Furthermore, a meta-analysis on MNS research on humans reports these same findings have been reported consistently in humans (Caspers et al., 2010). The implications of the MNS are vast, such that if the system is compromised due to abnormalities in the several structures that comprise the MNS, the results may contribute to early skill deficits in verbal behavior, such as echoic repertoires. The acquisition of echoic repertoires are posited to be integral to proceeding skill acquisition of more advanced verbal communication (e.g., DeSouza, Akers, & Fisher, 2017; Kisamore, Carr, & LeBlanc, 2011; Skinner, 1957; Sundberg & Michael, 2001), but can be difficult to teach to children with ASD (Drash et al., 1999; Esch et al., 2010; Shane, 2016). In summary, the STS has been associated with biological motion, emotional and social learning, and aiding in the development of verbal communication and theory of mind (Baron-Cohen et al., 1999; Frith, 2007; Gallagher et al., 2000; Hein & Knight, 2008; Zevin, 2009; Zilbovicius et al., 2006).

Although there is limited literature on the topic, previous studies examining grey matter (GM) volume in the STS have shown that when compared to TD counterparts, decreases in bilateral GM volumes of the STS are shown in Autistic children (N. Boddaert et al., 2004) and adults (Greimel et al., 2013; Hadjikhani et al., 2006). Similar findings have been reported in the left STS of Autistic adolescents and young adults (Chung et al., 2005). Conversely, studies with a sample of solely adolescents have found divergent results. Whilst one study reported an increased volume of the STS in

adolescents with ASD compared to TD adolescents (Waiter et al., 2004), another study found no difference (Hyde et al., 2010).

The other region of interest within the temporal lobe, namely the STG, is involved in analyzing facial expressions and gaze directions (Adolphs, 2009), as well as containing Wernicke's area; thus playing a role in the production and understanding of language (Bigler et al., 2007). Studies analyzing the volume development of the STG in Autistic adolescents have mixed findings. A range of studies reported increased volumes in Autistic participants in contrast to NT participants (Bonilha et al., 2008; Chung et al., 2005; Jou et al., 2010; Liu et al., 2017; X. Yang et al., 2016), whilst other studies report no differences (i.e., Bigler et al., 2007; Rojas et al., 2006) or decreased volumes in the ASD group (Cheng et al., 2011; Pereira et al., 2018). Notably, the Cheng and colleagues (2011) study mentioned also subdivided their ASD sample into an "Autism" and "Asperger" group. They report an increased volume of the STG in the "Asperger" sample in contrast to the "Autism" sample. However, they did not elucidate on their guidelines for creating these subgroups and did not compare them to their TD group. Therefore, it is unknown if their findings of lower volume may have been confounded by one subsample driving the discrepancy in volumes in their overall comparison ("Autism" + "Aspergers" vs TD). In addition to studies on full structure volume, other groups have conducted investigations on the cortical thickness (CT) of the STS across samples. Several studies on mixed age samples have found an overall thinner CT in Autistic individuals compared to TD counterparts (Patriquin et al., 2016; Wallace et al., 2010). A significant limitation of all these studies is that no study examined an adolescent-only sample. As not all studies controlled for age, and volume and cortical development of the STS and STG

tends to follow a curvilinear relationship (in this case, an upside-down U-shape) for both ASD and TD but with peak shifts (Greimel et al., 2013), age may have confounded their results. This is further supported by a recent finding of both STS and STG volume decreases found in an ASD sample compared to a TD sample, which were moderated by increasing age during adolescence to young adulthood (Pereira et al., 2018). Another limitation of these investigations which complicates a synthesis of the data is methods are not reported with a precision sufficient to deduce their units of measurement, and often present as examinations of the STS despite their results being related to the STG, or of the two areas' boundaries being operationally defined differently by different studies.

Prior studies examining the relation of social and communication challenges have reported associations between the STS and STG CT, with scores on the Social Responsiveness Scale (Prigge et al., 2018; Tu et al., 2016; Wallace et al., 2012), a questionnaire often used to quantify the severity of social challenges in Autistic individuals (Constantino, Przybeck, Friesen, & Todd, 2000). All three studies reported greater cortical thinness in either the STG (Tu et al., 2016; Wallace et al., 2012) or STS (Prigge et al., 2018) to be associated with greater SRS scores (i.e., greater severity of ASD). Additionally, bilateral volumes of STG have been reported to have a positive relation with the ADI-R social and communication total, whilst, conversely, a negative relation between the right STG and ADOS – Generic (Lord et al., 2000) score was reported (Rojas et al., 2006). Utilizing the Autism Quotient, a well-validated measure for screening social characteristics of ASD (Baron-Cohen, Wheelwright, Skinner, et al., 2001) in a NT sample, lower WM volumes, but not GM volumes, in the posterior STS were related to higher scores on the Autism Quotient (i.e., more characteristics of ASD;

von dem Hagen et al., 2011). Furthermore, as with the amygdala, STS and STG volumes were found to be related to challenges in identifying emotions from another person's eyes as assessed via the RMET (Sato et al., 2017). Specifically, volumes of the temporal parietal junction, an area which contains the STS and STG, had a negative correlation with the RMET score (i.e., greater volume was associated with lower/worse scores on the RMET) in their ASD sample. Interestingly, the opposite pattern emerged in their TD comparison sample (i.e., a positive association between the volume and RMET score). In conclusion, although results of both volume differences and volume relations with social and communication challenges are mixed and limited, it is important to examine changes in the STS and STG. This importance is highlighted by a prior study showing a causal relationship between activation of the STS and Amygdala which showed that when transcranial brain stimulation (TBS) was delivered to the right posterior STS, participants showed a decreased neural response to faces in the right posterior STS, and amygdala (Pitcher et al., 2017). Conversely, when the TBS was delivered to the vertex (i.e. the upper middle part of the head), no differences were observed in neural responses (Pitcher et al., 2017). Thus, it appears that further examination is warranted in STS and STG volumes in ASD to better understand the similarities and differences that exist across NT and neurodiverse samples in relations of these areas to social behavior and other social brain structures.

iii. Fusiform Gyrus. Residing ventrally on the temporal lobe and occipital lobe, the Fusiform Gyrus (FFG) has been implicated in detection and recognition of faces through differential activation to visual stimuli of neutral and emotional faces (e.g.,Iaria, Fox, Waite, Aharon, & Barton, 2008; Kanwisher, McDermott, & Chun, 1997; Kawasaki

et al., 2012; A. Puce, Allison, Gore, & McCarthy, 1995; Aina Puce, Allison, Asgari, Gore, & McCarthy, 1996; Zhang, Liu, & Xu, 2015).

A recent meta-analysis, utilizing data from 1,571 Autistic individuals and 1,651 NT individuals from across 49 sites, found that the CT of the FFG was significant thinner in ASD samples. Furthermore, the study reported that the FFG CT was the greatest during the period of adolescence (van Rooij et al., 2018). This finding is consistent with earlier investigations using adult comparison samples, which concluded that the right anterior FFG was significantly thinner in their ASD sample in contrast to their NT sample (Ecker et al., 2013; Hadjikhani et al., 2006). Additionally, Ecker et al., (2013) reported that their ASD sample showed increased surface area (SA) and decreased SA in the right and left FFG, respectively. Nonetheless, these findings are contrary to some earlier reports which showed increased CT in Autistic young adults (Khundrakpam et al., 2017), and Autistic adults (Hyde et al., 2010). In addition to CT studies, structural volume studies have shown increased GM volume (Bonilha et al., 2008; Rojas et al., 2006; Waiter et al., 2004), as well as decreased GM volumes of the FFG (Ha et al., 2015; Toal et al., 2010; Trontel et al., 2013) in Autistic samples in contrast to their NT counterparts. However, it should be noted that only two studies (i.e., Bonilha et al., 2008; Waiter et al., 2004) used an adolescent-only sample; both reported increased GM volumes of the FFG.

In regards to social and communication challenges, van Rooiji (2018) reported negative associations between CT of the FFG and ADOS scores, whilst an earlier study showed positive associations between the two factors (Khundrakpam et al., 2017). Despite the role of the FFG in the social brain, literature on the relation of FFG volume or CT to social behavior is limited. However, previous functional studies have highlighted

the importance of the FFG in social behavior (e.g., Bird, Catmur, Silani, Frith, & Frith, 2006; Dapretto et al., 2006; Ha et al., 2015; Hadjikhani et al., 2006; Itahashi et al., 2014; Kleinhans et al., 2008; Pierce et al., 2001; Richey et al., 2014; Scherf, Elbich, Minshew, & Behrmann, 2014). ASD findings, however, are mixed. Whilst some show hypoactivation (e.g., Dapretto et al., 2006; Pierce et al., 2001; Richey et al., 2014; Scherf, Luna, Minshew, & Behrmann, 2010) of the FFG during presentation of visual stimuli of faces, others show no abnormal activation (e.g., Bird, Catmur, Silani, Frith, & Frith, 2006; Hadjikhani et al., 2004; Kleinhans et al., 2008). Furthermore, negative links between FFG activation and SRS score (i.e., lower activation linked to greater social challenges) have been shown by at least one prior study (Scherf et al., 2014). Lastly, a recent study provided evidence for the link between functional and structural volume of the FFG. The study reported decreased GM volumes in the FFG, as well as decreased functional activity; additionally, the study reported links between GM volumes and the level of functional activity (Itahashi et al., 2014).

Taken together, previous research demonstrates that although results are mixed, differences in both CT and GM volumes of the FFG exist between Autistic and NT individuals (e.g., Bonilha et al., 2008; Ecker et al., 2013; Hadjikhani et al., 2006; Hyde et al., 2010; Khundrakpam et al., 2017; van Rooij et al., 2018; Waiter et al., 2004), and that these differences may be linked to social challenge severity (e.g., Itahashi et al., 2014; Khundrakpam et al., 2017; Scherf et al., 2014; van Rooij et al., 2018). Therefore, it is important to include the FFG when examining neurophysiological outcomes for Autistic individuals.

iv. Orbitofrontal Cortex. The Orbitofrontal Cortex (OFC) has been shown historically to support the social reinforcement and reward processes of stimuli-reinforcement associations (e.g., Rolls, 2000, 2004, 2006, 2019; Rolls, Critchley, Mason, & Wakeman, 1996; Thorpe, Rolls, & Maddison, 1983), with the medial OFC showing differential activation to visual stimuli of faces dependent on historical associations with reward (O'Doherty et al., 2003; Rolls et al., 1996; Thorpe et al., 1983). Furthermore, studies examining the effects of damage to the OFC in humans has shown greater challenges in recognition and prediction of others' negative affective responses (Blair & Cipolotti, 2000), and impaired production and recognition of emotional facial expressions (e.g., Damasio, Tranel, & Damasio, 1990; Hornak, Rolls, & Wade, 1996). Therefore, it has been hypothesized that a disfunction in the OFC may contribute to social challenges in ASD, stemming from inadequate stimuli-reinforcement association processing as related to social interactions (Bachevalier & Loveland, 2006; Baron-Cohen et al., 1999, 2000; Salmond et al., 2005).

Despite the hypothesized important role of the OFC in social reward value, the number of studies examining its volumetric abnormalities in ASD are few. Salmond and colleagues (2005) reported GM abnormalities in all but one of their Autistic adolescent participants when compared to a control sample of NT adolescents, but did not detail the type of abnormality. Later studies, albeit few in number, provided greater insight into abnormalities in the OFC found in Autistic samples. Decreases in both GM and white matter volumes of the right lateral OFC have been reported in children and adolescents with ASD (Girgis et al., 2007; Hardan et al., 2006). Additionally, increased GM volume was reported in the right inferior OFC in Autistic individuals relative to a NT sample,

although it should be noted the age range was vast (Range: 7- 29 years old; Hsiang-Yuan Lin, Hsing-Chang Ni, Meng-Chuan Lai, Wen-Yih Isaac Tseng, & Susan Shur-Fen Gau, 2015). Furthermore, differences became non-significant during a follow-up analysis using stratified ages (i.e., chidren, adolescents, and adults; Hsiang-Yuan Lin et al., 2015). A novel approach, at the time of publication, investigated structural differences in the OFC by comparing SA, CT, and cortical volume (a product of SA and CT). Using this approach, it was revealed that CT was increased in the right medial OFC, whilst SA was less in the lateral OFC for both hemispheres, in Autistic adults (Ecker et al., 2013). The finding of increased CT in the OFC was also supported by a recent meta-analysis showing consistent results across age development (van Rooij et al., 2018).

A number of the aforementioned studies examining abnormalities in the OFC of Autistic individuals have also examined associations of these abnormalities with social behavior (Ecker et al., 2013; Girgis et al., 2007; Hardan et al., 2006). However, due to the varied methodologies (e.g., GM volume versus cortical volume) and mixed results, it is difficult to discern consistency of outcomes. Whist all studies utilized the ADI-R in their correlational analyses, no associations were shown by Hardan et al., (2006), whilst negative correlations with OFC white matter volume (Girgis et al., 2007), and OFC cortical volume (Ecker et al., 2013) were found with the social domain of the ADI-R in later studies. Girgis and colleagues (2007) also reported the same directional correlation to be present for the social domain of the ADOS. Furthermore, one study, not aforementioned, which investigated adult samples, albeit not finding volumetric differences between their ASD and NT samples, found that both samples evidenced a negative relation between the AQ social impairment subscore and the right medial

Orbitofrontal gyrus volume. The importance of the OFC for social behavior related to challenges present in ASD are highlighted by the prior studies, yet further examination of these structural differences and relations to behavior is needed as part of intervention outcome literature utilizing biomarkers of intervention (Gebauer et al., 2015).

v. Insula. The insula, albeit not included in Brother's original Social Brain, has become a region of interest in relation to social communication due to its associations with affective processing of one's own private states (e.g., physical pain), as well as that of others, and empathy (e.g., Adolphs, 2009; A. D. Craig, 2002, 2008; Singer et al., 2004). For example, activation of the observer's insula has been shown when a painful shock is delivered to the hand of loved one (Singer et al., 2004).

Studies on Insular pathologies in ASD have revealed that Autistic adolescents show lower overall volumes in the right anterior insula and bilateral posterior insula when compared to NT adolescents (Parellada et al., 2017). Further investigation by Parellada et al. (2017) revealed that the difference in structure is also present in both GM volume and thickness of the posterior insula. These findings are consistent with a recent meta-analysis which showed decreased GM volume in the left posterior insula of Autistic participants compared to NT counterparts (Carlisi et al., 2017). Additional support for these findings are found in a prior report on young adults with pervasive developmental disorders, who also showed reductions in insular GM volumes (Kosaka et al., 2010). However, other studies show the opposite pattern, such as a meta-analysis which reported a higher likelihood of increased insular GM volume in children and adolescents with ASD (Bonilha et al., 2008; Duerden et al., 2012). Furthermore, a recent study of young adults with ASD examined SA, and found increased Insular SA compared to NT young

adults (Pereira et al., 2018). Although findings are mixed in the aforementioned reports, recent reports seem to indicate decreased GM volumes and increased SA in ASD.

In addition to structural differences, Parellada et al. (2017) report a negative relation between insular volumes and severity of both insight challenges (e.g., lack of judgement) and "Autistic-like" challenges (e.g., difficulties in social interaction and communication). This is consistent with reports that thinner cortex of the insula was associated with higher SRS scores (Tu et al., 2016), ADOS scores (van Rooij et al., 2018), SCQ scores, and RMET scores (Baribeau et al., 2019). No studies were found that reported a contradictory direction of relations; therefore it seems that although there is divergence in results of structural abnormalities across samples, results of the negative relation between social challenges and insular thickness are convergent. Hence, investigation into the relation of insular volume and thickness may provide greater insight into social skill intervention outcomes.

vi. Anterior Cingulate Cortex. The anterior cingulate cortex (ACC) is another subsequent candidate for structures of which the social brain is comprised because of its inherent location, sharing space with the limbic ("emotional") system and the prefrontal cortex ("cognitive") system (Stevens et al., 2011). Functional connectivity has been shown between the ACC and amygdala via *f*MRI when emotional stimuli are presented to participants, whereupon activation in the amygdala increases and subsequently decreases as the ACC increases in activation (Stevens et al., 2011). It has also shown a relation with attention and stimulus-reinforcer association (Bush et al., 2002; Pardo et al., 1990). As emotional recognition challenges (e.g.,Milosavljevic et al., 2016; Paula-Pérez, Martos-Pérez, & Llorente-Comí, 2010), and faulty stimulus control (e.g., Cengher et al.,

2015; L. Grow, Carr, Kodak, Jostad, & Kisamore, 2011; L. Grow & LeBlanc, 2013; Leaf et al., 2016) have been shown to be prevalent in ASD, the ACC has been investigated in recent years in relation to the neurobiology of ASD (Bonilha et al., 2008; Carlisi et al., 2017; Hadjikhani et al., 2004; Tu et al., 2016; van Rooij et al., 2018).

In studies of brain morphology comparing ASD and NT neuroanatomy, results have been mixed. Lower GM volume of the ACC was reported in Autistic individuals in a recent meta-analysis (Carlisi et al., 2017) and a subsequent study (Pereira et al., 2018), whilst other studies reported greater GM volume in ASD samples (Bonilha et al., 2008; Duerden et al., 2012; Toal et al., 2010), and no differences were found in an adolescent sample study (Tu et al., 2016). Furthermore, Periera and colleagues (2018) also reported increased SA and decreased CT in the ACC. Conversely, Van Rooiji and colleagues (2018) reported greater CT in the ACC for Autistic individuals

Studies on social and communication challenges have been shown in one identified ACC structural study, in which greater CT was associated with higher ADOS scores (van Rooij et al., 2018). In addition to the structural studies, the importance of the ACC in social behavior is elucidated by a study reporting lower functional connectivity between the ACC and subcortical areas as related to higher SRS social awareness subscale scores, while higher functional connectivity of the ACC and STG were also associated with worse scores on the subscale (Tu et al., 2016). Therefore, although research into the relation of structural ACC morphology and social behavior is limited, it is important to account for, considering its association with other important social brain areas (e.g., amygdala and STG).

These structures (i.e., amygdala, STS/STG, fusiform gyrus, OFC, and ACC), given their prior research findings in relation to social behavior and divergent morphology between ASD and NT individuals, have been shown to be important structures to examine when analyzing social skill intervention outcomes for Autistic individuals. Taken together, these structures may help elucidate the impact of social skills interventions on behavioral outcomes, as well as predictors of response to intervention.

B. The PEERS® Intervention

A well-established efficacious intervention for Autistic adolescents to develop and ameliorate their social skill challenges is the *Program for the Education and Enrichment of Relational Skills* ((PEERS®; e.g., Dolan et al., 2016; Frankel et al., 2010; Laugeson, Frankel, Gantman, Dillon, & Mogil, 2012; Laugeson, Frankel, Mogil, & Dillon, 2009; Rabin, Israel-Yaacov, Laugeson, Mor-Snir, & Golan, 2018; Schohl et al., 2014; Van Hecke et al., 2015; Yoo et al., 2014). Autistic individuals, aged 11 – 18, attend small-group weekly sessions for 14-weeks to work on making and keeping friends. The intervention involves an adolescent and caregiver group which run parallel to each other in separate rooms. The adolescents receive group instruction of didactic material, observe role-plays and practice perspective-taking at conclusion of role-play, complete behavioral rehearsal alongside a coach, and receive weekly homework assignments (Laugeson et al., 2009; Laugeson & Frankel, 2010b). Specific skills practiced include: identifying a common interest, developing two-way conversations, initiating and terminating a conversation, handling electronic communication, using appropriate humor, using good sportsmanship, and handling bullying. PEERS® has been replicated in the United States

outside the development site (Hill et al., 2017; Schohl et al., 2014) and five additional countries (i.e., Korea, Israel, China/Hong Kong, the Netherlands, and Canada), several of which have significantly different cultural norms than the development site at the University of California, Los Angeles (UCLA). Efficacy has been shown at seven additional sites (Hill et al., 2017; Jagersma et al., 2018; Marchica & D'Amico, 2016; Rabin et al., 2018; Schohl et al., 2014; Shum et al., 2019; Yoo et al., 2014). In addition to the social outcomes, completion of PEERS® has been related to significant reduction of depressive symptoms in adolescents (Schiltz et al., 2017), and social anxiety in adolescents (Hill et al., 2017; Schohl et al., 2014) and young adults (McVey et al., 2016). Furthermore, changes in EEG asymmetry, as well as changes in EEG coherence between different social brain regions, have been reported as neurophysiological outcomes of PEERS® (Haendel, 2018; Van Hecke et al., 2015). Thus, neurophysiological outcomes of PEERS® have previously been shown, and further examination into neurophysiological outcomes of PEERS® may enhance our understanding of outcomes and predictors of responders to the program. Furthermore, although prior neurophysiological outcomes utilizing EEG measures have been reported, no known study has examined outcomes utilizing MRI measures.

C. Summary and Aims of the Current Study

The purpose of the present study is to examine whether neurophysiological volumes of social brain structures change as a result of a social skills program randomized controlled trial, and whether neuroanatomical structural volumes and CT are predictors of questionnaire outcome measures, for the *PEERS*® intervention for Autistic

adolescents. The program was operated in a manner consistent with UCLA guidelines, using a randomized controlled trial and keeping fidelity with the *PEERS® for Adolescents* manual. The current study extends prior neurophysiological and outcome research in three important ways. First, social brain structures will be compared across an Experimental, Waitlist, and TD group of adolescents to examine significant differences in structure volumes and CT between groups at Pre- and Post-intervention. Second, structural regions involved in the social brain will be examined for change in volume or CT across the intervention. Third, structural volume and CT changes will be examined as predictors of positive outcomes measured via questionnaires. The first and foremost aim of this study is to examine neurophysiological outcomes utilizing structural MRIs and analyzing change in volume and CT in social brain regions over the course of the PEERS® intervention. Based on prior studies into the associations of these brain regions and previous reported outcomes of PEERS®, we hypothesized:

1) Structural volume, SA, and CT for all structures, save the Amygdala, will be significantly different at pre-intervention time between Autistic participants and NT counterparts. Due to amygdala volumes showing associations with several other mental health challenges (e.g., depression, anxiety, and PTSD; Hamilton et al., 2008; Herrington, Maddox, Kerns, et al., 2017; Morey et al., 2012), and the high comorbidity of ASD with these challenges (e.g., i.e., depression and anxiety; Simonoff et al., 2008), it is likely that amygdala volumes will be highly variable.
2) Structural volume, SA, and CT for all structures of the structures will show a change over the course of the PEERS® intervention in the experimental group with ASD that receives the intervention.

3) Structural volume changes will predict changes in questionnaire measures of social and communication deficits in ASD.

III. Method

A. Participants

Participants were enrolled in the current study utilizing a previously Institutional Review Board (IRB) approved advertisement and data collection (Schohl et al., 2014). Families that expressed interest in the program underwent a telephone-screening interview, administered by a graduate student in the clinical psychology doctoral program, to determine if the potential participant meets inclusion criteria. Inclusion criteria for Autistic participants was: 1) an assigned sex of male at birth; 2) adolescent participant with a chronological age between 12 years and 16 years old; 3) English fluency for the caregiver who was willing to participate in the study; 4) English fluency for the adolescent; 5) no prior history of an adolescent major mental illness (e.g., schizophrenia); 6) No metal implants or braces which precluded the adolescent participant from undergoing an MRI scan; 7) Successful completion of a mock scan; 8) no history of hearing, visual, or physical impairment which precludes the adolescent from participating in PEERS® activities or undergoing an MRI scan; 9) caregiver report of social difficulties in the adolescent participant; 10) An existing diagnosis of ASD, High Functioning ASD, Asperger's, or Pervasive Developmental Disorder – NOS; 11) Meeting criteria for ASD on the ADOS - G (Lord et al., 2000); 12) A minimum verbal IQ of 70 assessed by the Kaufmann Brief Intelligence Test – Second Edition (KBIT-2; Kaufman & Kaufman, 2004; Laugeson et al., 2009); 13) Adolescent expressed interest in

participating in a class (i.e., PEERS®) to help make and keep friends as assessed by the Adolescent Mental Status Checklist (Laugeson & Frankel, 2010b). Adolescents who attended intake and outtake (i.e., post-intervention test) visits were compensated $15 per hour in gift cards at the completion of their appointment. The PEERS® intervention was provided free of charge to families. Typically developing participants were only required to meet inclusion criteria 1 – 8, did not participate in the PEERS® intervention, and were also compensated $15 per hour in gift cards at the completion of their intake and outtake appointments.

Once potential participants with ASD were screened and were deemed to fit inclusion criteria, they were randomly assigned to the experimental (EXP) or waitlist (WL) group. Typically developing participants were placed in a Typically Developing (TD) group. The EXP group subsequently received the *PEERS® for Adolescents* intervention within two weeks of the intake appointment, whilst the WL participants did not receive the PEERS® treatment immediately. Outtake data were acquired following the completion of the 14th (i.e., final) session of the intervention for the EXP group, or within 14 weeks from intake for the WL and TD groups. Thereafter, the WL group received PEERS during the following session block (most commonly the next academic term, save the summer term). Using this parallel group design for groups allowed for examination of change over approximately 14 weeks and control for maturation effects. Table 1 shows a timeline of participant's stages of involvement.

Table 1

Treatment Timeline

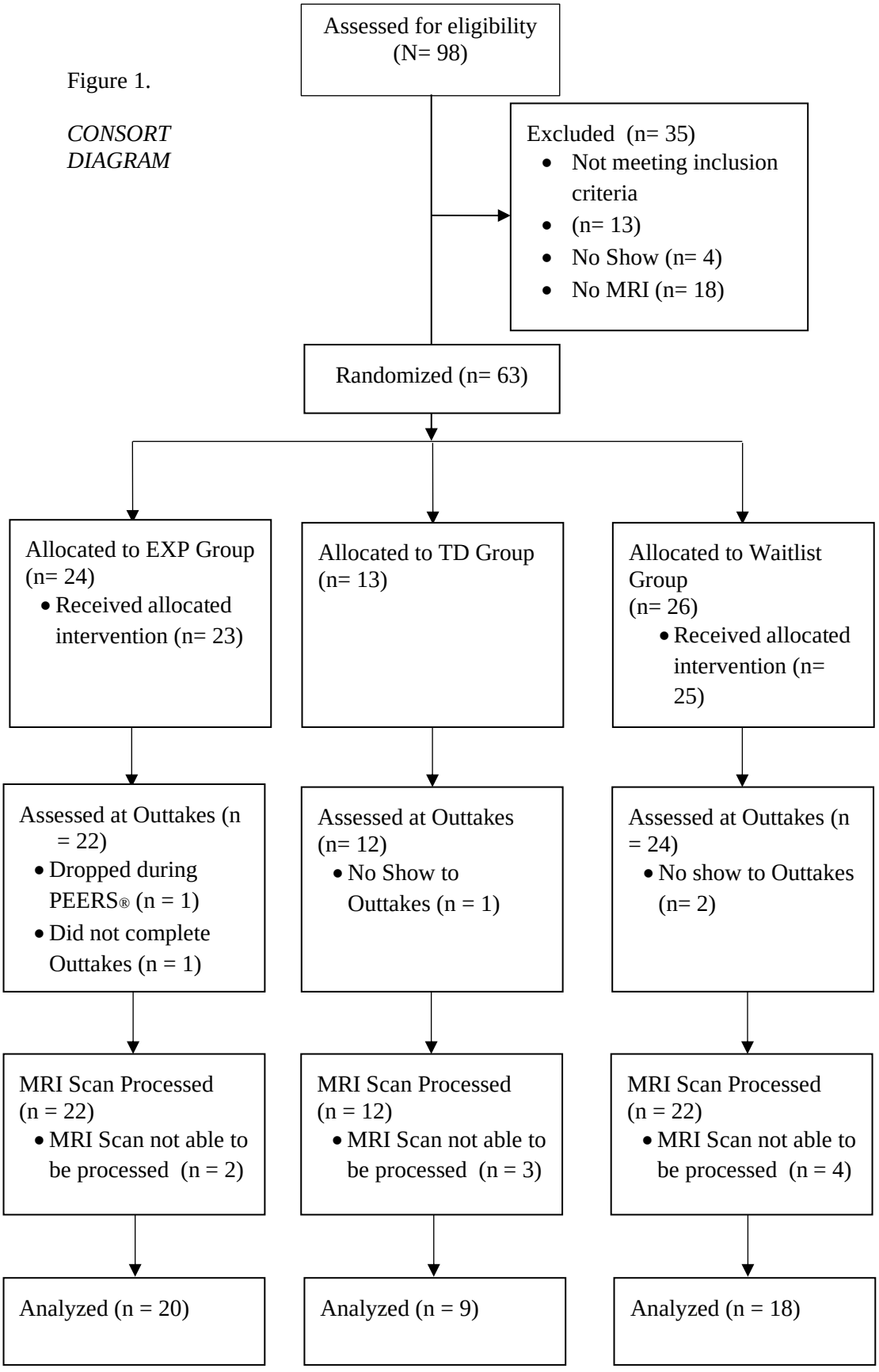
Group	Time 1	Treatment (14 Weeks)	Time 2	Treatment (14 weeks)
Experimental	Intakes	Receive PEERS®	Outtakes	
Waitlist	Intakes		Outtakes	Receive PEERS®
Typically Developing	Intakes		Outtakes	

B. Attrition.

Attrition was expected to approximate 20%, which is within the common range for randomized controlled trials (Hewitt et al., 2010). Additionally, participants who missed three or more sessions, or did not complete three homework assignments, were excused from the intervention and were not included in analyses. A consort diagram detailing participant recruitment, completion status, and data analysis status is found below on Figure 1.

Figure 1.

CONSORT DIAGRAM



C. Treatment

PEERS® for Adolescents was be delivered as in prior studies (Schohl et al., 2014). In brief, it was delivered in 90-minute parallel adolescent and parent/caregiver sessions on a weekly basis for 14 weeks. The PEERS® manual was adhered to for treatment. One graduate student or faculty with a Master's degree or higher and prior training in conducting the *PEERS® for Adolescents* intervention acted as the group leader for the adolescent group; another graduate student or faculty with prior training in *PEERS®* facilitated the caregiver group. Weekly supervision was conducted by the principal investigator, Dr. Van Hecke, who is certified by UCLA in providing the PEERS® models, as well as a licensed clinical psychologist collaborator, to insure adequate addressing of any mental health issues that might have arisen during the course of the intervention. Adolescents received points for appropriate participation, culminated with a graduation party and prizes at the final session.

Undergraduate research assistants in Dr. Van Hecke's lab, and interdisciplinary graduate students in speech pathology and audiology, trained and supervised by group leaders, acted as behavioral coaches, conducted role-plays, assisted with participant breaks, assisted with intake and outtake sessions, and assisted in adherence to fidelity of the intervention manual.

Adolescent PEERS® sessions started with a homework review from the previous week. Hereafter, a didactic lesson was provided by the group leader, role-plays demonstrating the incorrect versus correct manner in which to implement the skill for that week's didactic were conducted, and then a behavioral adolescent rehearsal followed, to practice the current skill being taught. Meanwhile, parent/caregiver sessions were

conducted in a similar manner without the rehearsal component. Weekly homework assignments were reviewed and difficulties in completing the assignments were addressed. A parent handout containing the curricular material of the adolescent session was given to parents and reviewed verbally by the parents/caregivers in conjunction with the caregiver group leader. Proactive troubleshooting of foreseeable difficulties in the skills to be practiced between the current and subsequent session occurred at each session.

Adolescents and parents/caregivers were reunified after the behavioral rehearsal for adolescents. Homework to be completed prior to the next session was assigned, and each adolescent and caregiver checked out with a team member prior to leaving the session.

D. Procedure

Participants attended two to three visits for their intake. Both Autistic groups, EXP and WL, attended one visit at Marquette University for screening and completion of questionnaires. Another visit took place at Froedtert Hospital Pavilion to undergo a mock MRI scan in a mock MRI scanner as an additional screen for inclusion criteria. A third visit at same location at Froedtert Hospital took place for the actual MRI scan. The mock scan and actual were separated by no more than two weeks' time between the two visits. Furthermore, the visit to Marquette University lasted approximately one to two hours; the mock scan 20 to 30 minutes; and the actual scan visit one to one and a half hours. The TD group completed the mock MRI scan and actual MRI scan in the same visit; thus only two visits were required in these cases. After intakes were completed, the EXP group

proceeded to receive PEERS®, whilst the WL and TD did not receive PEERS. After PEERS® was completed (no more than 16 weeks after intakes), participants from all three groups returned for outtakes. Outtakes were typically one visit. The appointment occurred at Froedtert Hospital Pavilion, where an MRI scan and questionnaires were completed. This visit lasted approximately one and a half to two hours.

E. Measures

i. Screening Measures. A demographics form and an adolescent health and medication history were completed at the intake appointments by all participants and their parent/caregiver. The subsequent forms were completed by adolescent participants. Adolescents had their cognitive functioning assessed via the KBIT-2. The resultant Verbal IQ was required to be a minimum of 70 to be included in the study. Adolescent interest in a program to make and teach friends was assessed using the Teen Mental Status Checklist (Laugeson & Frankel, 2010a). Diagnoses were confirmed utilizing the ADOS – G. The ADOS – G has served as the gold standard for assessment of ASD for almost two decades and has shown high validity, inter-rater reliability, and inter-item correlation (Lord et al., 2001). The ADOS-G was administered and scored by examiners trained to a research-level reliability within the lab. Inclusion criteria mandated a score indicating the presence of ASD. The EXP and WL group were compared for non-significant differences on the ADOS-G and KBIT-2 scores.

ii. Experimental Questionnaires: Parent/Caregiver-Report. Experimental measures were self-administered or read to the individual by a research assistant, when requested, at intake and outtake appointments. The following parent-report measures

utilized in our third aim (i.e., prediction of behavioral outcomes using brain structure volume and CT), with the exception of the Quality of Socialization Questionnaire and TASSK, which were only used to assess the efficacy of PEERS® in the current study, can be found below. All following questionnaires were repeated at intake and outtake.

1. ***Social Responsiveness Scale.*** The Social Responsiveness Scale 2 (SRS-2; Bruni, 2014; Constantino & Gruber, 2005) was administered to gauge the severity of social communication challenges. The SRS is a 65-item measure, which generates a raw total score, five subscale scores (Social Awareness, Social Cognition, Social Communication, Social Motivation, and Autistic Mannerisms), and Total T-Score, and is well validated (Bruni, 2014). Total T-scores between 60 and 75 indicate mild to moderate social difficulties, and Total T-Scores of 75 or higher are highly associated with a clinical diagnosis of ASD (Constantino & Gruber, 2005). Internal consistency for this study, measured using Cronbach's alpha (Cronbach, 1951) of the SRS subscales were as follows: a) Social Awareness = .25; b) Social Cognition = .67; c) Social Communication = .763; d) Social Motivation = .773; e) Autistic Mannerisms = .816. However, due to the small sample size of this study (n_{ASD} = 38), and the inherent larger sampling error found in these small samples (Javali et al., 2011), a Cronbach's alpha was computed for the total score as well as to increase the items included in the analysis (α = .91). All subscales were retained, as the total score provided an sufficient internal consistency score, and the current study being a pilot-study.

2. ***Quality of Socialization Questionnaire.*** The Quality of Socialization Questionnaire – Parent/Caregiver (QSQ-P; Laugeson et al., 2009) is a 12 item questionnaire which queries on the quantity of friends of the adolescent, frequency of adolescent get-together with peers, level of conflict, and number of friends involved in each get-together. The QSQ was administered to gauge the efficacy of PEERS in this study.
3. ***Child Behavioral Checklist.*** The Achenbach System of Empirically Based Assessment – Child Behavioral Checklist (CBCL; Achenbach & Rescorla, 2001) is a broadband measure to assess clinically elevated internalizing and externalizing symptoms, and contains eight DSM-oriented subscale scores that have been shown to be consistent with DSM-IV diagnostic criteria (Achenbach et al., 2002, 2003). It is comprised of 20 competence scores and 118 specific problem items; the latter was obtained for the current study. Raters score symptoms of their adolescent on a Likert scale ranging from 0 ("Not True") to 2 ("Very true"). Higher scores indicate greater severity of symptoms, with T-scores above 69 indicating clinically elevated symptoms (Achenbach & Rescorla, 2001). The subscale T-scores for Affective Problems, Anxiety Problems, Somatic Problems, Attention Deficit/Hyperactivity (ADH) Problems, Oppositional Defiant Problems, Conduct Problems, Obsessive-Compulsive Problems, and Post-Traumatic Stress Problems was utilized in this study. The CBCL DSM-Oriented subscale scores has been found to have an internal consistency range of .67 to .83, and have been validated for the assessment of emotional problems in youth with

ASD (Pandolfi et al., 2014). The internal consistency for this study were as follows: a) Affective Problems = . 74; b) Anxiety Problems = .60; c) Somatic Problems = .61; d) ADH Problems = .68; e) Oppositional Defiant Problems = .78; f) Conduct Problems = .78; g) Obsessive-Compulsive Problems = .56; h) Post-Traumatic Stress Problems = .71. A total DSM-5 Oriented Subscales internal consistency was also computed (α = .89) for the reasons abovementioned. All subscales were retained for analyses.

iii. Experimental Questionnaires: Self-Report. Questionnaires utilized to examine our third aim (i.e., behavioral outcome associations with brain structure morphology) was the Social Interaction Anxiety Scale (SIAS). Additionally, the Test of Adolescent Social Skills Knowledge was utilized to examine efficacy of the PEERS® intervention in this study.

1. ***Social Interaction Anxiety Scale.*** The Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998) measures anxiety while partaking in a social interaction. It queries anxieties such as being afraid to sound stupid, and not knowing what to say. The SIAS is scored using a Likert scale of agreement from 0 (not at all) to 4 (extremely), with higher scores indicating higher anxiety (Mattick & Clarke, 1998). Internal consistency in this study was good (α = .84).
2. ***Test of Adolescent Social Skills Knowledge.*** The Test of Adolescent Social Skills Knowledge (TASSK; Laugeson & Frankel, 2010) is a questionnaire designed to assess the knowledge directly targeted by the 13 didactic lessons of the PEERS® intervention. The questionnaire is comprised of 26 questions

(two questions per lesson), with higher scores reflecting greater knowledge of the social skills taught.

iv. Neuroimaging. Prior to their intake MRI scanning appointment, a mock scan was held whereupon the adolescent laid down in a simulated MRI machine, and the typical noise heard during an MRI scan was played. This was done to ensure increased likelihood of completion of the MRI procedure during intakes. At the time of intake, participants underwent structural, functional, and diffusion-weighted imaging, lasting about one hour. Only the structural MRIs (sMRI) were examined in the current study. Scans took place at Froedtert Hospital Pavilion in a research GE 3T scanner. An onsite Medical College of Wisconsin MRI technician administered the scans with assistance from the Van Hecke lab team.

1. ***Image Processing*.** Structural scans were processed using the automated image analysis program Freesurfer (version 6; http://surfer.nmr.mgh.harvard.edu/). The Freesurfer network is a well-validated tool for cortical parcellation and volumetric segmentation (subcortical structures) of brain scans to acquire the necessary data from MRI scans (Fischl, 2012) and is commonly used in brain research (e.g., Mikhael & Pernet, 2019; Pereira et al., 2018; van Rooij et al., 2018; D. Y.-J. Yang, Beam, Pelphrey, Abdullahi, & Jou, 2016). Freesurfer undergoes three main pre-processing auto-recon steps (see table 2 for detailed steps), in which the MRI scans acquired are converted from their raw scan to a more suitable format for analysis via 31 steps. The first autorecon (i.e., autorecon1) focuses on motion correction through skull stripping. The second (i.e., autorecon2) focuses on

subcortical segmentation through making the final surfaces. The third (i.e., autorecon3) focuses on spherical morphing and automatic cortical parcellation via the ASeg and Desikan-Killiany-Tourville (DKT) atlas. The first 26 steps are focused on transformation of the scan via processes such as motion correction, normalization (eliminates confounds in values due to different intensity in scans), skull stripping, registration (transformation of raw images to the correct orientation derived from a standard brain outline), and spherical mapping (i.e., labeling). The final steps focus on the parcellation in accordance to the DKT atlas, which is recommended as the best practice approach to cortical parcellation in comparison to other processing tools (Mikhael & Pernet, 2019). Cortical parcellation also is processed through the ASeg (Auto-Segmented subcortical structures) atlas. For the purpose of this study, also utilized the Destrieux atlas (a2009) as it results in the parcellation and segmentation of gyri and sulci of brain regions (Destrieux et al., 2010), such as the Superior Temporal lobe which results in the STS and STG. MRI scans that cannot be processed through Freesurfer were excluded from analysis as it is indicatory of excess movement from the participant during the scan to a degree that Freesurfer can no longer apply sufficient motion correction or an accurate registration. Data was retrieved from Freesurfer after processing and analysis of scans and imported to SPSS 26.0 (*IBM SPSS Statistics for Mac*, 2019) for analysis.

Table 2
Freesurfer Autorecon Processing Stages

Autorecon 1	Autorecon 2	Autorecon 3
1. Motion Correction	6. Linear Volumetric Registration	24. Spherical Mapping
2. Non-Uniform Intensity Normalization	7. CA Intensity Normalization	25. Spherical Registration
3. Talairach transform computation	8. CA Non-linear	26. Spherical Registration of the contralateral hemisphere
4. Intensity Normalization 1	9. Remove Neck	27. Map average curvature of subject
5. Skull Strip	10. LTA with Skull	28. Cortical Parcellation - DKT (Labeling)
	11. CA Label	29. Cortical Parcellation Statistics
	12. Intensity Normalization 2	30. Cortical Ribbon Mask
	13. White Matter (WM) Segmentation	31. Cortical Parcellation mapping to ASeg
	14. Edit WM with ASeg	
	15. Fill	
	16. Tessellation	
	17. Smooth1	
	18. Inflate1	
	19. QShpere	
	20. Automatic Topology Fixer	
	21. Final Surfs	
	22. Smooth 2	
	23. Inflate2	

F. Data Analytic Plan

An alpha level of .05 was used for significance criterion for hypothesis tests. One-way ANOVAs were utilized to explore group differences between the EXP, WL, and TD on demographic variables and total brain volume. Additionally, group differences on the ADOS were explored using a t-test for EXP versus WL groups. We hypothesized that there would not be group differences on these variables.

To examine the first aim, Pearson's bivariate correlation were run on measures of volume to examine the extent of collinearity between the structures of close proximity (e.g., STS and STG), and structures for which the total structure morphology measures (e.g., FFG volume) and subsegments of that structure (e.g., lateral FGG) to determine if fewer Multivariate Analyses of Variance (MANOVAs), thus containing more dependent variables, was better suited for analysis as recommended in the use of multivariate statistics for neuroscience (Carey, 2013). Thus, the number of MANOVAs was dictated by the significant correlations found across structures. Multivariate analyses of variance were used to examine whether the dependent variable (DV) of volumes for each structure and hemisphere (e.g., left amygdala and right amygdala volume, or left Insula and right Insula CT) varied by Group (EXP versus WL versus TD). MANOVAs were conducted to examine grey matter volume in the following structures: a) amygdala; b) STG; c) STS d) rostral and caudal anterior cingulate (rACC and cACC) e) ACC gyri and sulci f) mACC gyri and sulci) g) lateral and medial OFC; h) Lateral FFG i) FFG; j) Insula. Following this, another set of MANOVAs was utilized to examine bilateral cortical SA as the dependent variable, for the above listed *b – i*. Lastly, another set of MANOVAs examined bilateral CT as the dependent variable, for structures labeled *b – i*.

To evaluate the second aim, Omnibus Group (EXP versus WL versus TD) x Time (intake versus outtake) mixed methods, repeated-measures multivariate analysis of variance (MANOVAs) were conducted. Measures included were the same as those conducted in the aforementioned MANOVA examination for group differences at pre-intervention; one set of mixed methods MANOVAs for volumes of the ten structures/areas as the DV, a separate set of mixed methods MANOVAs for the ten

Cortical Thicknesses (CTs) as the DV, and a separate set of mix methods MANOVAs for the ten surface areas (SAs)as the DV.

Lastly, to evaluate the third aim, difference scores were first calculated for each structure and its DVs (volume, CT, and SA) that showed a significant change in the second aim. Following this computation, an Omnibus Group (EXP versus WL versus TD) x Time (intake versus outtake) mixed method repeated-measures multivariate analysis of covariance (MANCOVA) was conducted. Measures included were the SRS-2, SIAS and CBCL as DVs. Difference scores for change over time in a structure morphology were included as a covariate. A separate mix methods MANCOVA was conducted for each significant finding from the second aim. Thus, the number of mixed methods MANOVAs were dictated by the number of significant results from aim 2. Significant findings were explored utilizing follow up ANCOVAs and *post-hoc* simple effect tests at the univariate level.

IV. Results

A. Data Screening

Data were screened for normality, outliers, and impossible values.

i. Behavioral Data. Behavioral Data revealed the following: a) One subject was missing the entirety of his post-test behavioral data; he was thus excluded from behavioral analyses, but retained for imaging analyses; b) five outliers were identified (0.7% of total behavioral data), one on the CBCL Anxiety Problems subscale, one on the CBCL Somatic Problems subscale, two on the CBCL Obsessive Compulsive Problems subscale, and one on the CBCL Post-Traumatic Stress Problems subscale.

As none of the outliers identified were extreme, and a multivariate analysis of variance (MANOVA) indicated that there were no significant differences across pre-test scores on the CBCL subscales scores, data analyses proceeded without changes to the outliers.

Therefore, data analyses for aim 3 were processed including the 5 outlier points, and compared to results from data analyses with these points excluded. If the removal of the outliers did not change significance, they were retained in the presented results. All behavioral data was within normal limits of skew. Kurtosis was not within normal limits but was due to the small sample size of each group.

ii. Imaging Data. Data were screened for each structure and morphology of the structure (Volume, SA, and CT). Therefore, for any outliers found in the structural data, the data analyses were processed including and excluding the outlying data points for that structure. If there were no differences in the significance of the statistical outcomes after exclusion, compared to inclusion, data were retained in those analyses to retain power. If outlier inclusion versus exclusion affected the significance of the results, data were excluded or replaced using Winsorization to the next highest or second lowest value (Tabachnick & Fidell, 2013). .

iii. Group Differences. An ANOVA and an independent sample T-test were utilized to assess EXP versus WL versus TD group differences on demographic variables and total brain volume. As hypothesized, no significant differences were uncovered for age ($F(2,44) = .531$, $p = .592$), KBIT-2 Verbal IQ ($F(2,44) = .1.914$, $p = .160$), KBIT-2 Nonverbal IQ ($F(2,44) = .030$, $p = .971$), KBIT-2 Full Scale IQ ($F(2,44) = ..834$, $p = .441$), ADOS-G total scores ($t(36) = -.926$, $p = .361$), and total brain volume

($F(2,44) = .233$, $p = .793$). Table 3 shows demographic variables and total brain volume for all groups.

Table 3

Demographics for Experimental, Waitlist, and Typically Developing groups at pre-test

	Group (N = 47)			
	Experimental (n = 20) $M(SD)$	Waitlist Control (n = 18) $M(SD)$	Typically Developing (n = 9) $M(SD)$	p
Age (years)	13.50 (1.40)	13.56 (1.82)	14.11 (1.17)	*ns*
KBIT-2 Verbal IQ	96.85 (19.50)	107.00 (19.59)	108.22 (11.57)	*ns*
KBIT-2 Nonverbal IQ	103.60 (17.56)	104.72 (14.85)	104.89 (17.12)	*ns*
KBIT-2 Full Scale IQ	100.45 (19.47)	107.17 (18.14)	107.67 (14.16)	*ns*
ADOS-G Total Score	12.50 (3.91)	(13.83 (4.95)	N/A	*ns*
Total Brain Volume (mm^3)	1303882.25 (110925.01)	1325642.50 (157808.96)	1334899.55 (88269.96)	*ns*
TASSK[a]	12.15 (3.10)	21.2(3.55)	N/A	*ns*
QSQ – P (%)[a]				*ns*
0 Friends	40	10	N/A	
1 Friend	20	15	N/A	
2 Friends	15	0	N/A	
3 Friends	5	0	N/A	
4 Friends	5	35	N/A	
5 Friends	5	20	N/A	
6 -8 Friends	10	20	N/A	

[a]The following variables had a different n value: Waitlist (n = 17), and Experimental (n = 20)

Furthermore, as abovementioned, multivariate analysis of variance (MANOVA) indicated that there were no significant differences across EXP and WL groups on pre-test scores on the CBCL subscales (affective problems, anxiety problems, somatic problems, attention deficit/hyperactivity problems, somatic problems, oppositional defiant problems, conduct problems, obsessive compulsive problems, and post-traumatic stress problems; Wilk's Lambda = .679, $F(8,28) = 1.652$, $p = .155$). A MANOVA further revealed a significant difference present in SRS subscale and total scores at the pre-test across EXP versus WL group (Wilk's Lambda = .694, $F(5,31) = 2.730$, $p = .037$). Univariate follow-ups showed a significant difference in the SRS Cognition Subscale score ($F(1,32) = 4.499$, $p = .041$). However, this significance did not survive a Bonferroni correction (alpha = .008), thus no further action was required. Independent sample t-tests for comparison of pre-test differences were not significant for either the SIAS ($t(35) = -.335$, $p = .740$) or the TASSK ($t(35) = -1.446$, $p = .157$). Two missing items were missing from the QSQ-P total get-togethers. Data were analyzed for patterns using multiple imputation pattern analysis and data was found to be missing at random. A multiple imputation (five iterations) was subsequently conducted to impute values for those data points (0.135% of data; Tabachnick & Fidell, 2013). A chi-square of independence revealed no significant differences between EXP versus WL groups in total get-togethers at the pre-test χ_2 (7, N = 37) = 3.953, $p = .785$, Cramer's V = .327).

B. PEERS® Efficacy

A repeated-measures ANOVA was utilized to examine change over time between EXP and WL groups on the TASSK. Results revealed a significant time x group

interaction ($F(2, 43) = 43.340$, $p < .001$, Partial $\eta^2 = .683$). Follow-up paired sample t-tests showed a significant increase in scores (i.e., greater social skill knowledge) from pre-test to post-test for the EXP group ($M = 21.20$, $SD = 3.55$; $t(19) = -11.092$, $p < .001$, Cohen's $d = 2.480$), whilst the WL group showed no change over time ($M = 15.16$, $SD = 3.14$; $t(16) = -1.846$, $p = .083$, Cohen's $d = .449$). Furthermore, a chi-square test of independence showed a significant difference between groups at post-test χ^2 (8, N = 37) = 3.953, $p = .016$, Cramer's V = .711), with parents reporting more adolescent get-togethers in the EXP group than in the WL group, after reports initially showed no difference in this measure at pre-test as described in "Group Differences."

V. Structure Volume (Aim 1 and 2)

A. Amygdala

i. Screening. Four outliers (2.13% of data) were identified in amygdala volumes, three (one extreme and two mild) in the left amygdala, and one in the right amygdala. One extreme outlier was subsequently Winsorized to the next highest value (Tabachnick & Fidell, 2013). The subsequent analyses were found to not be affected by the inclusion versus exclusion of the remaining outliers, thus data points were retained.

ii. Aim 1 (Group Differences). A MANOVA at pre-test revealed no significant differences between groups (i.e., EXP versus WL versus TD; Wilk's Lambda = .876, $F(4,86) = 1.465$, $p = .220$) across left and right amygdalae. Thus, our hypothesis, that amygdala volumes between Autistic participants (i.e., EXP and WL) versus TD participants at pre-test would not be significantly different, was supported.

iii. Aim 2. A multivariate group x time interaction was found to be significant (Wilk's Lambda = .528, $F(4,86) = 8.054$, $p < .0001$, Partial $\eta^2 = .273$). Univariate tests showed the interaction to be significant for both left ($F(2,44) = 12.228$, $p = .00006$, Partial $\eta^2 = .357$) and right amygdalae ($F(2,44) = 10.830$, $p = .00015$, Partial $\eta^2 = .330$). A follow-up paired sample t-tests across groups from pre- to post-test revealed the following: a) In the EXP group, a significant decrease in volume from pre- to post-test for in the left ($t(19) = 4.272$, $p = .0004$), and right amygdalae ($t(19) = 4.272$, $p = .0007$); b) In the WL and TD, a significant increase in volume in the right, and left amygdala, respectively. However, neither significance for the WL or TD group findings survived a Bonferroni correction. Refer to table 4 for means and standard deviations. The results of repeated-measures MANOVA supported the prediction that significant volume changes would be detected in the EXP group compared to the WL and TD group.

Table 4

Means and Standard Deviations for Group by Time Interaction of Amygdala Volumes (mm^3) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Amygdala							<.0001
Left Amygdala	1970.52 (229.18)	1839.13 (227.04)	1857.00 (295.52)	1860.47 (304.24)	1895.53 (251.46)	1863.25 (242.80)	<.0001
Right Amygdala	2049.10 (200.91)	1939.74 (182.90)	1944.41 (265.37)	2019.88 (269.37)	1998.41 (233.31)	1996.85 (229.95)	<.001

B. Superior Temporal Gyrus (STG), and Superior Temporal Sulcus (STS)

i. Screening. Due to the proximity of the STG and STS, a Pearson's bivariate correlation was run to examine if these two structures showed high collinearity, thus supporting a single, instead of two separate, MANOVAs. Prior to this analysis, two outliers (0.53% of data) were identified, one in the left STS and one the left STG. Both outliers were resulting from the same participant, with the STG outlier being an extreme outlier. Therefore, the extreme outlier was subsequently Winsorized to the second lowest value (Tabachnick & Fidell, 2013). Subsequent analyses showed no difference in results regardless of whether one or both outliers were trimmed, Winsorized or not transformed. Therefore, the presented analysis included a retained mild outlier and transformed extreme outlier. Pearson's correlations at pre-test revealed strong significant correlations between the Left STG and Left STS ($r(45) = .606$, $p < .00001$), Left STG and Right STS ($r(45) = .601$, $p < .00001$), Left STS and Right STS ($r(45) = .675$, $p < .000001$). Furthermore, the Right STG was not significantly correlated with the Right STS ($r(45) = -.285$, $p = .052$), Left STG ($r(45) = -.068$, $p = .650$), or Left STS ($r(45) = -0.125$, $p = .401$). Given the strong correlations found otherwise, a single model was run with all four variables.

ii. Aim 1 (Group Differences). An omnibus MANOVA revealed, no significant differences between the three groups across any of the structures (Wilk's Lambda = .967, $F(4,42) = .354$, $p = .839$). Contrary to our hypothesis, there were not group differences between the autistic participants and their TD counterparts at the pre-time point.

iii. Aim 2. An omnibus repeated-measures MANOVA revealed a multivariate significant main effect of time (Wilk's Lambda = .533, $F(4,41) = 8.978$, $p < .0001$,

Partial η^2 = .467), but not a significant group by time interaction. Univariate tests showed that the multivariate main effect was driven by a significant change in the left STG volume across time points ($F(1,44) = 9.373$, p =.004, Partial η^2 = .176). A follow-up paired sample t-test showed a significant increase over time in left STG volumes for the WL group ($t(17) = -3.182$, p = .005), and TD group ($t(19) = -3.426$, p = .009), whilst no change was shown in the EXP group ($t(19) = -1.814$, p = .086). Both WL and TD group remained significant after a Bonferroni correction (Bonferroni et al., 1936). Therefore, our hypothesis for Aim 2 that, in the EXP group, STS and STG volumes would change over the course of the PEERS® intervention was not supported. Refer to table 5 for means and standard deviations.

Table 5

Means and Standard Deviations for Group by Time Interaction of STG and STS Volumes (mm^3) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
STG and STS							.743
Left STG	19525.05 (3860.59)	21266.80 (3179.22)	20542.06 (1760.11)	21719.61 (2672.14)	19466.89 (2889.96)	20867.83 (1364.94)	.847
Right STG	19675.85 (2586.88)	20228.05 (3102.54)	20964.17 (2963.43)	20015.16 (1995.36)	19667.33 (2278.06)	19867.67 (1968.97)	.417
Left STS	12219.60 (1631.02)	12170.25 (1431.62)	12227.61 (1938.99)	11950.44 (2016.90)	11941.33 (2024.87)	12015.78 (1748.80)	.629
Right STS	13248.20 (1508.20)	13286.75 (1809.61)	13551.06 (1620.31)	13214.00 (1523.02)	13634.00 (1804.72)	13625.78 (1342.12)	.449

C. Anterior Cingulate Cortices (ACC gyri and sulci; middle AAC gyri and sulci, rostral ACC, caudal ACC)

i. Screening. Due to both subsegmented measures of the full ACC (e.g., rostral and caudal ACC subsegments), and the overall ACC (i.e., ACC gyri and sulci) being examined, Pearson's bivariate correlation were run to examine the extent of collinearity between the structures, to determine if a fewer MANOVAs (containing more dependent variables) was better suited for analysis as recommended in the use of multivariate statistics for neuroscience (Carey, 2013). Prior to this analysis, twenty-two unique outliers (2.92% of data) were identified, two in the left ACC, seven in the right ACC, three in the left middle ACC (mACC), two in the right mACC, two in the left caudal ACC (cACC), one in the right cACC, three in the left rostral ACC (rACC), and three in the right rACC. As the latter 3 structures (i.e., mAAC, cACC, and rACC) are subsegements of the overall ACC, many of the outlying points were resulting from the same participants found to be outliers in the overall ACC, thus only unique outliers are presented in the total outlier count. Two extreme outliers were detected in the left mACC and Winsorized to the next highest value (Tabachnick & Fidell, 2013). Subsequent analyses showed no difference in results regardless of whether the remaining mild outliers were trimmed, Winsorized or not transformed. Therefore, the presented analysis included a retained mild outlier and transformed extreme outlier.

Pearson's correlations at pre-test revealed strong significant correlations within hemispheres across structures and not across hemispheres. Therefore, subsequent analyses were run using structures within a hemisphere for a total of two MANOVAs for

Aim 1, and two repeated-measures MANOVA for hypothesis 2. Refer to table 6 for correlation matrix.

Table 6

Correlations between bilateral ACC, mACC, cACC, and rACC at pre-test (N = 47)

Variables	1	2	3	4	5	6	7	8
1. Left ACC	-	.729**	.653**	.576**	.325*	.039	.587**	-.122
2. Right ACC	.729**	-	.679**	.470**	.176	.063	.608**	.071
3. Left mACC	.653**	.679**	-	.650**	.227	-.002	.435**	-.076
4. Right mACC	.576**	.470**	650**	-	.473**	-.057	.365*	-.113
5. Left cACC	.325*	.176	.227	.473**	-	-.210	.460**	-.051
6. Right cACC	.039	.063	-.002	-.057	-.210	-	.025	.447**
7. Left rACC	.587**	.608**	.435**	.365*	.460**	.025	-	.012
8. Right rACC	-.122	.071	-.076	-.113	-.051	.447**	.012	-

*$p < .05$ **$p < .01$

ii. Aim 1 (Group Differences). Two omnibus MANOVAs revealed, contrary to our hypothesis, that there were not group differences between any of our groups in structures (i.e., ACC, mACC, rACC, cACC) for either the left hemisphere (Wilk's Lambda = .834, $F(8,82) = .972$, $p = .464$), or right hemisphere (Wilk's Lambda = .854, $F(8,82) = .843$, $p = .568$) at pre-test.

iii. Aim 2. An omnibus repeated-measures MANOVA for the right hemisphere structures showed a Box's test of equality of covariance matrices was violated (Box's M = 156.84, $F(72, 2103.419) = 1.453$, $p = .008$), therefore Pillai's Trace was used.

No significant effect at the multivariate level was found for either a main effect of time (Pillai's Trace = .055, $F(4,41) = .593$, $p = .670$) or an interaction effect (Pillai's Trace = .190, $F(8, 84) = 1.101$, $p = .352$).

The left hemisphere structures revealed a multivariate significant main effect of time (Wilk's Lambda = .262, $F(4,41) = 8.978$, $p < .00001$, Partial $\eta^2 = .738$), but not a significant group by time interaction (Wilk's Lambda = .786, $F(8,82) = 1.313$, $p = .249$, Partial $\eta^2 = .114$). Univariate tests showed that the multivariate main effect was driven by a significant change in the left cACC volume ($F(1,44) = 56.416$, $p < .000001$, Partial $\eta^2 = .562$) and left rACC ($F(1,44) = 95.250$, $p < .000001$, Partial $\eta^2 = .684$) across time points. Additionally, a univariate test showed a time x group interaction for the left cACC ($F(2,44) = 5.120$, $p = .01$, Partial $\eta^2 = .189$). The interaction was further explored with paired t-tests which showed a significant increase in volume in the EXP ($t(19) = -6.76$, $p < .00001$, Cohen's $d = 1.493$), WL ($t(17) = -4.939$, $p < .001$, Cohen's $d = 1.165$), and TD ($t(8) = -4.231$, $p = .003$, Cohen's $d = 1.410$). All groups remained significant following a Bonferroni-Holm correction (Holm, 1979). To further examine the difference in change over time between WL and EXP group, a repeated-measures ANOVA was run with the left cACC as the sole dependent variable. The repeated-measures ANOVA revealed that the EXP group showed a change greater than that of the WL group ($F(1,36) = 4.153$, $p = .049$, Partial $\eta^2 = .103$) Therefore, our hypothesis for aim 2 was partially supported by the time by group interaction found in volume change of the left cACC. However, the remainder of the structures did not support our hypothesis. Refer to table 7 for means and standard deviations.

Table 7

Means and Standard Deviations for Group by Time Interaction of ACC Volumes (mm^3) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Left Hemisphere							.249
Left ACC	5831.45 (853.75)	6012.00 (698.11)	5915.94 (872.50)	5960.33 (871.64)	5207.89 (808.96)	5355.33 (881.85)	.776
Left mACC	3031.85 (533.99)	3114.50 (577.19)	3070.28 (537.90)	3272.56 (675.40)	2818.22 (347.08)	2895.56 (213.41)	.710
Left cACC	2853.80 (429.84)	4142.05 (670.47)	3062.28 (787.34)	3836.78 (593.69)	3100.11 (430.80)	3533.67 (471.17)	.010
Left rACC	3171.25 (649.88)	4663.20 (738.26)	3395.72 (991.47)	4574.33 (930.50)	3134.44 (763.52)	4260.11 (615.69)	.409
Right Hemisphere							.371
Right ACC	7323.30 (1094.87)	7610.95 (1173.91)	7966.67 (995.65)	7691.11 (1266.78)	7229.44 (967.66)	7461.33 (1219.80)	.033
Right mACC	3331.35 (439.25)	3321.35 (387.44)	3401.67 (581.17)	3324.00 (532.60)	3331.56 (574.89)	3346.56 (488.65)	.786
Right cACC	2727.50 (510.48)	2877.70 (396.48)	2804.78 (487.25)	2809.28 (759.62)	2914.22 (499.47)	3106.00 (486.97)	.805
Right rACC	3063.90 (674.16)	3264.15 (635.63)	3322.33 (694.84)	3001.72 (714.70)	3129.56 (861.97)	3131.00 (777.27)	.225

D. Lateral and Middle Orbitofrontal Cortex (lOFC and mOFC)

i. Screening. Due to measures (i.e., lOFC and mOFC) forming the full OFC, Pearson's bivariate correlation were run to examine the extent of collinearity between the structures, to determine the number of MANOVAs to be used. Prior to this analysis, thirteen outliers (3.46% of data) were identified. No outlier was extreme and removing the outlier did not change the results, therefore they were retained without transformation. Pearson's correlations at pre-test revealed strong significant correlations within hemispheres across structures (i.e., lOFC and mOFC) and not across hemispheres. Therefore, subsequent analyses were run using structures within a hemisphere for a total of two MANOVAs for hypothesis 1, and two repeated-measures MANOVA for hypothesis 2. Refer to table 8 for correlation matrix.

Table 8

Correlations between bilateral OFC and lateral OFC at pre-test (N = 47)

Variables	1	2	3	4
1. Left lateral OFC	-	.140	.698**	.062
2. Right lateral OFC	.140	-	.122	.794**
3. Left mOFC	.698**	.140	-	.021
4. Right mOFC	.062	.794**	.021	-

*p < .05 **p <.01

ii. Aim 1 (Group Differences). Two omnibus MANOVAs revealed, contrary to our hypothesis, groups were not significantly different in volume within the left hemisphere (Wilk's Lambda = .923, $F(4,86) = .880$, $p = .479$) and within the right hemisphere (Pillai's Trace = .082, $F(4,88) = .935$, $p = .449$) at pre-test. Therefore, there were not group differences between our autistic participants and their TD counterparts at the pre-time point for any of the structures.

iii. Aim 2. An omnibus repeated-measures MANOVA for the left hemisphere structures showed a significant main effect of time at the multivariate level (Wilk's Lambda = .717, $F(2, 43) = 8.473$, $p < .001$, Partial $\eta^2 = .229$) and no significant interaction effect (Wilk's Lambda = .900, $F(4, 86) = 1.159$, $p = .335$). Univariate tests revealed the significant main effect of time was driven by the mOFC ($F(2, 43) = 8.473$, $p < .001$, Partial $\eta^2 = .229$). A follow-up paired sample t-test showed that only the EXP showed a significant increase in mOFC volume from pre-test to post-test ($t(19) = -3.321$, $p = .004$, Cohen's $d = 0.743$), and remained significant after a Bonferroni Correction. There was no significant difference across time for the WL group ($t(17) = -1.890$, $p = .076$), or TD group ($t(8) = -1.489$, $p = .175$).

An omnibus repeated-measures MANOVA for the right hemisphere structures indicated that Box's test of equality of covariance matrices was violated (Box's M = 58.818, $F(20, 2574.123) = 2.468$, $p < .001$), therefore Pillai's Trace is utilized in subsequent results. No significant main effect of Time (Pillai's Trace = .009, $F(4, 43) = .193$, $p = .825$) or interaction effect of Time x Group (Pillai's Trace = .038, $F(4, 88) = .430$, $p = .786$) was found. Refer to table 9 for means and standard deviations.

Therefore, our hypothesis that change in OFC volumes over the course of PEERS® in the EXP group was not supported as no time by group interaction effect was found.

Table 9

Means and Standard Deviations for Group by Time Interaction of OFC Volumes (mm^3) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Left							.335
Lateral OFC	11278.10 (1635.80)	11497.30 (1937.93)	12160.44 (1618.48)	11598.61 (1548.58)	11479.44 (1299.73)	11815.22 (1092.38)	.228
Medial OFC	5247.30 (609.78)	5971.10 (768.96)	5589.89 (889.36)	5916.33 (811.36)	5194.33 (846.48)	5574.67 (689.48)	.335
Right							.786
Lateral OFC	11322.20 (1852.35)	11414.15 (1858.72)	11335.22 (1423.38)	11524.89 (1727.31)	11495.22 (1385.07)	11495.44 (789.51)	.976
Medial OFC	5462.45 (643.96)	5359.10 (750.38)	5189.06 (770.88)	5420.78 (626.49)	5308.22 (1008.00)	5489.67 (509.69)	.625

E. Fusiform Gyrus (FFG sulci and gyri, and lateral FFG)

i. Screening. Due to the lateral FFG being a subsegment of the full segmented FFG volume (i.e., FFG sulci and gyri), Pearson's bivariate correlations were run to examine the extent of collinearity between the structures, to determine the number of MANOVAs to be used. Prior to this analysis, seven outliers (1.86% of data) were identified, three in the left FFG, three in the right FFG, and one in the right lateral FFG. Three of the seven outliers were extreme outliers, one in the left FFG, one in the right FFG, and one in the right lateral FFG were Winsorized to the second lowest value (Tabachnick & Fidell, 2013). Subsequent analyses showed no difference in results regardless if the remaining mild outliers were trimmed or not transformed. Therefore, the remaining mild outliers were retained in the analyses. Pearson's correlations at pre-test revealed strong significant correlations across structures and hemispheres, with the exception of the right FFG which showed no correlation to other structures (i.e., left FFG, right lateral FFG, and left lateral FFG). Therefore, a single MANOVA with all four structures as dependent variables per analysis was utilized. Refer to table 10 for correlation matrix.

Table 10

Correlations between bilateral FFG and lateral FFG at pre-test (N = 47)

Variables	1	2	3	4
1. Left lateral FFG	-	.659**	.674**	.248
2. Right lateral FFG	.659**	-	.838**	.186
3. Left FFG	.674**	.838**	-	.122
4. Right FFG	.248	.186	.122	-

*$p < .05$ **$p < .01$

ii. Aim 1 (Group Differences). A MANOVA at pre-test indicated that Box's Test of Equaity of Covariance was violated (Box's M = 37.70, $F(72, 2574.123) = 1.582$, $p = .048$), therefore Pillai's Trace is presented. Our hypothesis of significant differences at pre-test in FFG volumes was not supported as no significant differences between groups (i.e., EXP versus WL versus TD) in any of the four structures across time (Pillai's Trace = .259, $F(8, 84) = 1.561$, $p = .149$) was found.

iii. Aim 2. An omnibus repeated-measures MANOVA for the left hemisphere structures indicated that Box's Test of Equality of Covariance was violated (Box's M = 155.46, $F(72, 2103.419) = 1.440$, $p = .010$), and thus, Pillai's Trace is subsequently presented. No significant main effect of time at the multivariate level (Pillai's Trace = .124, $F(4, 41) = 1.453$, $p = .234$), and no significant interaction effect (Pillai's Trace = .311, $F(8, 84) = 1.936$, $p = .065$) were found. The null hypothesis, therefore, was not able to be rejected, and our hypothesis was not supported. Refer to table 11 for means and standard deviations.

Table 11

Means and Standard Deviations for Group by Time Interaction of Fusiform Gyri Volumes (mm^3) *for all groups at Pre- and Post-Test*

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Fusiform Gyrus (FFG)							.065
Right FFG	10954.60 (1451.30)	10308.85 (1378.69)	10593.06 (1211.52)	10821.89 (1364.69)	10704.56 (2133.51)	11768.22 (745.50)	.054
Left FFG	10156.60 (1491.50)	10410.05 (1409.49)	11212.89 (1243.08)	10939.11 (1357.64)	11600.00 (732.76)	10992.22 (1096.17)	.154
Right Lateral FFG	5965.50 (943.81)	6069.10 (1037.22)	6370.06 (1104.97)	6179.00 (1146.96)	6708.78 (1047.59)	6968.78 (612.80)	.122
Left Lateral FFG	5367.85 (701.96)	5515.70 (852.24)	5809.33 (1028.40)	5687.50 (930.05)	5809.33 (1258.27)	5816.67 (1135.96)	.824

F. Insula

i. Screening. One extreme outlier (0.53%) were identified in insula volumes. This value neared an impossible value as it was more than five standard deviations from the mean. The outlier was subsequently Winsorized to the next highest value (Tabachnick & Fidell, 2013).

ii. Aim 1 (Group Differences). A MANOVA with both left Insula, and right Insula as dependent variables, at pre-test revealed no significant differences between groups (i.e., EXP versus WL versus TD; Wilk's Lambda = .876, $F(4,86) = 1.488$, $p = .213$). Our hypothesis, that insula volumes between Autistic participants (i.e., EXP and WL) versus TD participants at pre-test would be significantly different, was not supported.

iii. Aim 2. An omnibus mix models repeated-measures MANOVA showed no significant main effect of time at the multivariate level (Wilk's Lambda = .953, $F(2, 43) = 1.069$, $p = .352$), and no significant interaction effect (Wilk's Lambda = .822, $F(4, 86) = 2.211$, $p = .074$). Refer to table 12 for means and standard deviations. Our hypothesis, that insula volumes would significantly change in the EXP group from pre- to post-PEERS® ,was not supported.

Table 12

Means and Standard Deviations for Group by Time Interaction of Insula Volumes for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre *M*(*SD*)	Post *M*(*SD*)	Pre *M*(*SD*)	Post *M*(*SD*)	Pre *M*(*SD*)	Post *M*(*SD*)	
Insula							.074
Left Insula (mm^3)	7427.35 (814.52)	7323.55 (749.59)	7730.72 (890.61)	7388.83 (956.13)	7150.78 (942.32)	7333.78 (790.00)	.021
Right Insula (mm^3)	7574.85 (805.77)	7492.75 (883.80)	8010.56 (946.55)	7656.17 (842.16)	7399.67 (1049.08)	7429.44 (966.55)	.700

VI. Structure Volume and Behavioral Measures (Aim 3)

Mixed method, repeated-measures multivariate analysis of covariance (MANCOVAs) between groups (EXP versus WL versus TD), across pre- to post-test on CBCL subscales (i.e., Affective Problems, Anxiety Problems, Somatic Problems, Attention Deficit/Hyperactivity Problems, Oppositional Defiant Problems, Conduct Problems, and Post-Traumatic Stress Problems), revealed that, contrary to the hypothesis, there was no interaction between changes in behavioral scores and

structure changes. Structures which showed a significant time by group interaction (i.e., Left Amygdala, Right Amygdala, Left STG and Left ACC) were entered as covariates into the model. Results indicated Box's Test of Equality of Covariance was violated (Box's M = 147.540, $F(136, 3569.532) = 1.220$, $p = .045$), therefore Pillai's Trace was used. No significant interactions between CBCL subscale scores across time, and difference scores for each structure entered as a covariate were found at the multivariate level (Left Amygdala: Pillai's Trace = .288, $F(8,24) = 0.884$ $p = .544$; Right Amygdala: Pillai's Trace = .254, $F(8,24) = 1.023$ $p = .446$; Left STG: Pillai's Trace = .189, $F(8,24) = 0.700$ $p = .688$; Left cACC: Pillai's Trace = .247, $F(8,24) = 0.982$ $p = .474$).

An additional MANCOVA for the SRS subscales and SIAS scores, revealed that, contrary to the hypothesis, there was no interaction between changes in behavioral scores and structure changes in social behavior outcomes. No significant interactions between SRS subscale scores and SIAS total score across time, and difference scores for each structure entered as a covariate were found at the multivariate level (Left Amygdala: Wilk's Lambda = .806, $F(6,26) = 1.043$, $p = .421$; Right Amygdala: Wilk's Lambda = .879, $F(6,26) = 0.598$, $p = .729$; Left STG: Wilk's Lambda = .942, $F(6,26) = 0.267$, $p = .947$; Left cACC: Wilk's Lambda = .853, $F(6,26) = 0.748$, $p = .616$). Our hypothesis that Therefore, our hypothesis for Aim 3, that structural volume changes would predict changes in behavioral measures, was not supported. Refer to table 13 for means and standard deviations.

Table 13

Means and Standard Deviations for Time by Structure Difference Scores for EXP and WL groups at Pre- and Post-Test on Behavioral Measures

	Group (N = 46)[a]							
	Experimental (n = 20)		Waitlist Control (n = 17)					p
	Pre M(SD)	Post M(SD)	Pre M(SD)	Post M(SD)	Right Amygdala	Left Amygdala	Left STG	Left cACC
CBCL					.544	.446	.688	.474
Affective Problems	65.15 (9.91)	62.00 (10.74)	65.12 (7.88)	63.24 (9.44)	.950	.406	.212	.828
Anxiety Problems	65.80 (9.20)	62.80 (8.58)	67.35 (5.38)	64.41 (8.45)	.318	.086	.454	.512
Somatic Problems	56.75 (8.64)	56.40 (8.09)	59.06 (6.97)	56.00 (7.37)	.631	.777	.411	.524
ADH[b] Problems	60.45 (5.90)	59.55 (6.10)	63.06 (6.69)	59.76 (7.84)	.435	.994	.330	.206
Oppositional Defiant Problems	57.90 (7.28)	57.70 (8.60)	59.35 (8.37)	58.59 (9.43)	.969	.457	.666	.042

Conduct Problems	58.10 (7.28)	56.80 (6.67)	56.29 (7.40)	55.29 (7.24)	.072	.890	.468	.116
Obsessive-Compulsive Problems	68.95 (8.00)	65.15 (9.46)	66.71 (9.90)	64.76 (8.70)	.151	.681	.696	.598
Post-Traumatic Stress Problems	64.65 (7.67)	62.60 (8.79)	67.71 (7.40)	64.53 (6.83)	.442	.810	.487	.573
SRS Subscales & SIAS					.729	.421	.947	.616
Social Awareness	12.00 (3.08)	10.40 (3.41)	13.35 (2.64)	12.53 (3.84)	.854	.815	.538	.826
Social Cognition	21.65 (5.82)	17.30 (5.23)	18.24 (3.46)	16.94 (3.99)	.904	.510	.305	.410
Social Communication	34.75 (7.45)	29.65 (7.88)	38.41 (8.70)	34.71 (9.83)	.835	.261	.418	.665
Social Motivation	16.10 (6.47)	13.05 (5.87)	17.76 (4.93)	15.53 (4.82)	.272	.587	.574	.962
Autistic Mannerisms	20.45 (6.48)	16.55 (5.85)	20.06 (6.18)	17.65 (6.12)	.530	.708	.775	.680
SIAS total score	31.95 (10.47)	24.65 (10.87)	33.24 (12.88)	32.88 (12.06)	.296	.377	.997	.387

[a]*N* = 46 due to one WL participant not returning his outtake forms, [b]ADH = Attention Deficit/Hyperactivity Problems; *N = 46*

VII. Structure Surface Area (Aim 1 and 2)

A. Superior Temporal Gyrus (STG), and Superior Temporal Sulcus (STS)

i. Screening. Due to the results of the abovementioned correlations in structure volume, a single MANOVA was conducted. Prior to this analysis, fourteen outliers (3.70% of data) were identified, seven in the left STS, two in the right STS, two in the left STG and two the right STG. Two of the fourteen outliers discovered were extreme outliers, one in the left STS and one in the right STG. Therefore, the extreme outliers were subsequently Winsorized, one to the next highest value and the other outlier to the second lowest value (Tabachnick & Fidell, 2013). Subsequent analyses showed no difference in results regardless of whether the remaining outliers were trimmed, Winsorized or not transformed. Therefore, the presented analysis retained mild outliers and one transformed extreme outlier. Furthermore, because of the correlations aforementioned in the volume section between STS and STG, a single model was run with all four variables.

ii. Aim 1 (Group Differences). An omnibus MANOVA revealed, no significant differences between the three groups across any of the structures (Wilk's Lambda = .912, $F(8,84) = .485$, $p = .856$). Contrary our hypothesis, there were not group differences between our autistic participants and their TD counterparts at the pre-time point.

iii. Aim 2. An omnibus repeated-measures MANOVA for the left hemisphere structures revealed no significant main effect of time (Pillai's Trace = .054, $F(4,41) = .587$, $p = .674$) on SA. Furthermore, no significant group by time interaction on SA was revealed by the MANOVA (Pillai's Trace = .054, $F(8, 84) = .290$, $p = .968$).

Therefore, the hypothesis that the EXP group would show a change in SA from pre-test to post-test was not supported by the results. Refer to table 14 for means and standard deviations.

Table 14

Means and Standard Deviations for Group by Time Interaction of STG and STS Surface Area (mm^2) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
STG and STS							.968
Left STG	5975.20 (659.81)	5946.75 (671.95)	6056.67 (783.47)	6002.17 (759.47)	5770.44 (503.28)	5864.44 (372.70)	.475
Right STG	5512.60 (570.74)	5498.60 (626.73)	5486.94 (546.22)	5429.44 (583.71)	5403.11 (515.70)	5450.44 (376.29)	.353
Left STS	4726.05 (569.73)	4642.10 (471.23)	4606.00 (658.01)	4527.50 (714.14)	4543.22 (657.30)	4579.33 (553.04)	.501
Right STS	5084.40 (736.67)	5088.55 (601.42)	5055.17 (524.86)	4974.83 (587.00)	5079.44 (477.16)	5079.22 (376.29)	.339

B. Anterior Cingulate Cortices (ACC gyri and sulci; middle AAC gyri and sulci, rostral ACC, caudal ACC)

i. Screening. Eight unique outliers (0.90% of data) were identified, three in the left mACC, one in the right mACC, two in the left cACC , two in the left rACC, and one in the right rACC. Subsequent analyses showed no difference in results regardless if the mild outliers were trimmed, Winsorized or not transformed. Therefore, the presented analysis included a retained mild outlier and transformed extreme outlier.

A total of two MANOVAs for hypothesis 1, and two repeated-measures MANOVAs for hypothesis 2 were conducted for reasons elucidated in the volume results section for the ACC, in reference to correlation findings across subsegments of the ACC.

ii. Aim 1 (Group Differences). Two omnibus MANOVAs revealed, contrary to our hypothesis, that there were not group differences between any of our groups in structures (i.e., ACC, mACC, rACC, cACC) for either the left hemisphere (Wilk's Lambda = .839, $F(8,82) = .942$, $p = .487$), or right hemisphere (Wilk's Lambda = .872, $F(8,82) = .726$, $p = .668$).

iii. Aim 2. An omnibus repeated-measures MANOVA for the left hemisphere structures indicated that Box's test of equality of covariance matrices was violated (Box's M = 147.540, $F(72, 2103.419) = 1.367$, $p = .023$), therefore Pillai's Trace was used. No significant effect at the multivariate level was found for either a main effect of time (Pillai's Trace = .070, $F(4,41) = .774$, $p = .549$) or an interaction effect (Pillai's Trace = .158, $F(8, 84) = .899$, $p = .521$).

The left hemisphere structures also indicated that Box's test of equality of covariance matrices was violated (Box's M = 177.341, $F(72, 2103.419) = 1.643$, $p =$

.001), therefore Pillai's Trace was used. No significant effect at the multivariate level was found for either a main effect of time (Pillai's Trace = .020, $F(4,41) = .205$, $p = .934$) or an interaction effect (Pillai's Trace = .218, $F(8, 84) = 1.282$, $p = .264$). Therefore, the hypothesis that the EXP group would show a change in SA from pre-test to post-test was not supported by the results. Refer to table 15 for means and standard deviations.

Table 15

Means and Standard Deviations for Group by Time Interaction of STG and STS Surface Area (mm^2) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre *M*(*SD*)	Post *M*(*SD*)	Pre *M*(*SD*)	Post *M*(*SD*)	Pre *M*(*SD*)	Post *M*(*SD*)	
Left Hemisphere							.521
Left ACC	1787.15 (230.79)	1823.45 (226.13)	1812.22 (260.99)	1782.78 (289.02)	1583.44 (199.28)	1655.11 (208.62)	.235
Left mACC	1007.40 (149.01)	1043.10 (159.18)	995.50 (180.01)	987.72 (177.09)	907.44 (114.07)	929.33 (86.51)	.275
Left cACC	1263.80 (183.92)	1308.70 (185.30)	1226.67 (179.84)	1213.67 (193.89)	1128.11 (117.48)	1143.00 (109.61)	.328
Left rACC	1378.65 (204.46)	1391.05 (236.48)	1399.56 (241.21)	1349.28 (288.35)	1245.22 (153.45)	1310.11 (164.56)	.699
Right Hemisphere							.264
Right ACC	2275.00 (294.16)	2326.80 (346.88)	2416.39 (364.27)	2304.39 (392.25)	2253.67 (235.12)	2302.44 (306.09)	.234
Right mACC	1030.80 (112.30)	1040.90 (104.02)	1069.67 (174.95)	1048.89 (170.23)	1065.78 (199.72)	1091.11 (237.64)	.444
Right cACC	890.00 (136.64)	902.30 (114.95)	897.22 (215.58)	870.83 (231.84)	970.56 (105.66)	978.11 (119.96)	.023
Right rACC	920.45 (174.82)	927.40 (198.70)	913.72 (205.64)	863.39 (187.14)	934.78 (151.59)	945.11 (154.76)	.410

C. Lateral and Middle Orbitofrontal Cortex (lOFC and mOFC)

i. Screening. Ten outliers (2.66% of data) were identified. No outlier was extreme and removing the outliers did not change the results, therefore they were retained without transformation. Subsequent analyses were run using structures within a hemisphere for a total of two MANOVAs for hypothesis 1, and two repeated-measures MANOVA for hypothesis 2.

ii. Aim 1 (Group Differences). Two omnibus MANOVAs revealed, contrary our hypothesis, groups were not significantly different in SA within the left hemisphere (Wilk's Lambda = .895, $F(4,86) = 1.224$, $p = .307$) or within the right hemisphere (Wilk's Lambda = .973, $F(4,86) = .298$, $p = .878$) . Therefore, there were not group differences between our autistic participants and their TD counterparts at the pre-time point for any of the of structures.

iii. Aim 2. An omnibus repeated-measures MANOVA for the left hemisphere structures showed no significant main effect of time at the multivariate level (Wilk's Lambda = .976, $F(2, 43) = .536$, $p = .589$), as well as no significant time x group interaction effect (Wilk's Lambda = .894, $F(4, 86) = 1.245$, $p = .298$). Therefore, the hypothesis that the EXP group would show a change in SA from pre-test to post-test was not supported by the results.

An omnibus repeated-measures MANOVA for the right hemisphere structures showed no significant main effect of time at the multivariate level (Wilk's Lambda = .080, $F(2, 43) = 1.875$, $p = .166$), as well as no significant time x group interaction effect (Wilk's Lambda = .949, $F(4, 86) = .573$, $p = .683$). Therefore, the hypothesis that the

EXP group would show a change in SA from pre-test to post-test was not supported by the results. Refer to table 16 for means and standard deviations.

Table 16

Means and Standard Deviations for Group by Time Interaction of OFC Surface Area (mm^2)for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Left							.298
Lateral OFC	3653.30 (621.07)	3549.75 (544.61)	3664.61 (414.59)	3648.22 (544.61)	3546.67 (427.05)	3704.67 (317.65)	.409
Medial OFC	1783.00 (202.20)	1814.30 (259.38)	1878.56 (209.54)	1848.61 (209.79)	1753.44 (174.66)	1829.89 (88.90)	.334
Right							.683
Lateral OFC	3930.75 (523.25)	3740.05 (601.91)	3937.22 (517.35)	3788.67 (462.30)	3743.33 (519.86)	3774.67 (390.36)	.341
Medial OFC	1715.30 (147.82)	1673.85 (255.39)	1696.61 (187.70)	1663.89 (189.28)	1678.11 (257.13)	1732.00 (222.90)	.538

D. Fusiform Gyrus (FFG sulci and gyri, and lateral FFG)

i. Screening. Eight outliers (2.13% of data) were identified, five in the right FFG, and seven in the right lateral FFG. Two of the eight outliers were extreme outliers, both in the right FFG. One was Winsorized to the second lowest value; one was Winsorized to the next highest value (Tabachnick & Fidell, 2013). Subsequent analyses showed no difference in results regardless if the remaining mild outliers were trimmed, or not transformed. A single MANOVA with all four structures as dependent variables per analysis was utilized.

ii. Aim 1 (Group Differences). A MANOVA at pre-test revealed, contrary to the hypothesis, no significant differences between groups (i.e., EXP versus WL versus TD) in any of the four structures across time (Wilk's Lambda = .870, $F(8, 82) = .739$, $p = .657$).

iii. Aim 2. An omnibus mixed methods repeated-measures MANOVA showed no significant main effect of time at the multivariate level (Wilk's Lambda = .962, $F(4, 41) = .400$, $p = .807$), and no significant interaction effect (Wilk's Lambda = .876, $F(8, 82) = .699$, $p = .691$). Therefore, the hypothesis that the EXP group would show a change in SA from pre-test to post-test was not supported by the results. Refer to table 17 for means and standard deviations.

Table 17

Means and Standard Deviations for Group by Time Interaction of Fusiform Gyri Surface Area (mm^2) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Fusiform Gyrus (FFG)							.691
Right FFG	3226.55 (394.12)	3204.45 (392.86)	3478.61 (534.42)	3410.11 (458.53)	3496.44 (561.72)	3577.56 (469.32)	.434
Left FFG	3280.65 (354.75)	3230.90 (346.21)	3402.17 (439.28)	3357.61 (402.08)	3386.33 (557.36)	3428.00 (453.41)	.606
Right Lateral FFG	1562.65 (225.78)	1504.95 (207.04)	1636.89 (358.53)	1605.94 (364.56)	1696.33 (310.76)	1749.78 (138.98)	.200
Left Lateral FFG	1411.20 (179.02)	1383.65 (204.43)	1445.22 (228.61)	1427.83 (210.30)	1477.44 (297.97)	1471.89 (292.65)	.897

E. Insula

i. Screening. Two outliers (1.06%) were identified in insula volumes, both in the right Insula. One extreme outlier was subsequently Winsorized to the second lowest value (Tabachnick & Fidell, 2013). Subsequent analyses showed no difference in results regardless if the remaining mild outlier was trimmed, or not transformed.

ii. Aim 1 (Group Differences). A MANOVA with both left and right Insula as dependent variables at pre-test revealed no significant differences between groups at the multivariate (Pillai's Trace = .027, $F(4, 88) = .301$, $p = .877$). Our hypothesis, that insula volumes between Autistic participants (i.e., EXP and WL) versus TD participants at pre-test would be significantly different, was not supported.

iii. Aim 2. An omnibus mixed model repeated-measures MANOVA showed no significant main effect of time at the multivariate level (Pillai's Trace = .010, $F(2, 43) = .208$, $p = .813$), and no significant interaction effect (Pillai's Trace = .125, $F(4, 88) = 1.471$, $p = .218$). Therefore, the hypothesis that the EXP group would show a change in SA from pre-test to post-test was not supported by the results . Refer to table 18 for means and standard deviations.

VIII. Structure Surface Area and Behavioral Measures (Aim 3)

As no significant changes in SA were found for any of the structures, Aim 3 was not applicable to SA of these structures.

Table 18

Means and Standard Deviations for Group by Time Interaction of Insula Surface Area (mm^2) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Insula							.218
Left Insula	2156.05 (196.15)	2115.45 (188.73)	2145.39 (309.27)	2144.89 (307.69)	2103.78 (216.82)	2123.22 (207.99)	.078
Right Insula	2238.80 (234.15)	2219.15 (265.29)	2257.67 (276.10)	2238.61 (266.89)	2172.78 (241.66)	2196.00 (214.50)	.604

IX. Structure Cortical Thickness (Aim 1 and 2)

A. Superior Temporal Gyrus (STG), and Superior Temporal Sulcus (STS)

i. Screening. Because of results of the abovementioned correlations in structure volume, a single MANOVA per analysis was conducted. Nine outliers (2.39% of data) were identified, three in the left STS, two in the right STS, two in the

left STG and two the right STG. Three of the nine outliers discovered were extreme outliers, two in the left STS and one in the right STG. Therefore, the extreme outliers were subsequently winsorized, two to the next highest value and the one to the second lowest value (Tabachnick & Fidell, 2013). Subsequent analyses showed no difference in results regardless of whether the remaining outliers were trimmed, Winsorized or not transformed. Therefore, the presented analysis included retained mild outliers and transformed extreme outlier.

ii. Aim 1 (Group Differences). An omnibus mixed methods MANOVA revealed, no significant differences on cortical thickness (CT) between the three groups across any of the structures (Wilk's Lambda = .722, $F(8, 84) = 1.854$, $p = .079$). Contrary to our hypothesis for Aim 1, there were not group differences between our autistic participants and their TD counterparts at the pre-time point.

iii. Aim 2. An omnibus mixed methods repeated-measures MANOVA indicated no significant main effect of time (Wilk's Lambda = .916 , $F(4, 41) = .938$, $p = .452$) on CT. Furthermore, no significant group by time interaction on CT was revealed by the MANOVA (Wilk's Lambda = .758 , $F(8, 84) = 1.520$, $p = .163$) on CT. Therefore, the hypothesis for Aim 2, that the EXP group would show a change in thickness from pre-test to post-test was not supported by the results. Refer to table 19 for means and standard deviations.

Table 19

Means and Standard Deviations for Group by Time Interaction of STG and STS cortical thickness (mm) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
STG and STS							.163
Left STG	2.99 (0.15)	2.99 (0.16)	3.07 (0.12)	3.03 (0.13)	2.97 (0.09)	3.01 (0.12)	.281
Right STG	3.05 (0.12)	3.06 (0.16)	3.12 (0.14)	3.09 (0.16)	2.99 (0.09)	3.05 (0.13)	.085
Left STS	2.72 (0.15)	2.74 (0.13)	2.78 (0.08)	2.78 (0.11)	2.78 (0.07)	2.75 (0.08)	.385
Right STS	2.78 (0.18)	2.81 (0.14)	2.86 (0.15)	2.86 (0.14)	2.83 (0.13)	2.84 (0.14)	.315

B. Anterior Cingulate Cortices (ACC gyri and sulci; middle AAC gyri and sulci, rostral ACC, caudal ACC)

i. Screening. Fifteen unique outliers (1.99% of data) were identified, one in the left ACC, six in the right ACC, five in right mACC, two in the left cACC, and one in the right cACC. Subsequent analyses showed no difference in results regardless if the remaining mild outliers were trimmed, Winsorized or not transformed. Therefore, the presented analysis included a retained mild outlier and transformed extreme outlier.

A total of two MANOVAs for hypothesis 1, and two repeated-measures MANOVAs for hypothesis 2 were conducted for reasons elucidated in the volume results section for the ACC, in reference to correlation findings across subsegments of the ACC.

ii. Aim 1 (Group Differences). Two omnibus MANOVAs revealed, contrary our hypothesis, there were not group differences between any of our groups in CT of structures (i.e., ACC, mACC, rACC, cACC) for the left hemisphere (Wilk's Lambda = .810, $F(8, 84) = 1.164$, $p = .330$). The MANOVA containing the right hemisphere indicated that Box's test of equality of covariance matrices was violated (Box's M = 42.576, $F(20, 2551.423) = 1.790$, $p = .017$), therefore Pillai's Trace was used. A multivariate analysis revealed no significant differences between groups in structures of the right hemisphere (Pillai's Trace = .247, $F(8, 86) = 1.518$, $p = .163$).

iii. Aim 2. An omnibus repeated-measures MANOVA for the left hemisphere structures indicated that Box's test of equality of covariance matrices was violated (Box's M = 181.580, $F(72, 2088.827) = 1.691$, $p < .001$), therefore Pillai's Trace was used. No significant effect at the multivariate level was found for either a main effect of time (Pillai's Trace = .029, $F(4, 42) = .317$, $p = .865$) or an interaction effect (Pillai's Trace = .190, $F(8, 86) = 1.129$, $p = .352$).

An omnibus repeated-measures MANOVA for the right hemisphere structures also indicated that Box's test of equality of covariance matrices was violated (Box's M = 156.255, $F(72, 2088.827) = 1.455$, $p = .008$), therefore Pillai's Trace was used. No significant effect at the multivariate level was found for a main effect of time (Pillai's Trace = .148, $F(4, 42) = 1.830$, $p = .141$). Furthermore, a significant time x group interaction effect at the multivariate level was uncovered (Pillai's Trace =

.326, $F(8, 86) = 2.097$, $p = .045$). However, follow-up univariate tests showed no significant interactions for any of the structures: ACC ($F(2, 45) = .398$, $p = .674$); mACC ($F(2, 45) = 2.145$, $p = .129$); cACC ($F(2, 45) = 1.955$, $p = .153$); rACC ($F(2, 45) = 1.524$, $p = .229$). Therefore, the hypothesis that the EXP group would show a change in CT from pre-test to post-test was not supported by the results. Refer to table 20 for means and standard deviations.

Table 20

Means and Standard Deviations for Group by Time Interaction of STG and STS cortical thickness (mm) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Left Hemisphere							.352
Left ACC	2.97 (0.15)	3.02 (0.12)	3.04 (0.13)	3.08 (0.20)	3.01 (0.10)	2.94 (0.17)	.078
Left mACC	2.94 (0.19)	2.94 (0.11)	3.02 (0.07)	3.01 (0.14)	2.99 (0.14)	2.96 (0.12)	.876
Left cACC	2.89 (0.18)	2.89 (0.16)	2.96 (0.13)	2.98 (0.17)	2.87 (0.20)	2.84 (0.22)	.617
Left rACC	3.00 (0.16)	3.03 (0.15)	2.95 (0.19)	3.05 (0.24)	2.99 (0.17)	2.89 (0.11)	.067
Right Hemisphere							.045
Right ACC	2.86 (0.15)	2.88 (0.12)	2.95 (0.11)	2.95 (0.16)	2.84 (0.12)	2.88 (0.14)	.153
Right mACC	2.91 (0.11)	2.93 (0.10)	2.98 (0.11)	2.98 (0.13)	2.84 (0.05)	2.96 (0.10)	.229
Right cACC	2.65 (0.14)	2.66 (0.20)	2.69 (0.22)	2.75 (0.27)	2.65 (0.19)	2.62 (0.18)	.674
Right rACC	3.01 (0.25)	3.04 (0.20)	3.06 (0.14)	3.01 (0.19)	2.94 (0.12)	3.00 (0.14)	.129

C. Lateral and Middle Orbitofrontal Cortex (lOFC and mOFC)

i. Screening. Twenty outliers (5.32% of data) were identified, nine in the left lOFC, three in the right lOFC, one in the left mOFC, and six in the right mOFC. No outlier was extreme and removing the outliers did not change the results, therefore they were retained without transformation. Subsequent analyses were run using structures within a hemisphere for a total of two MANOVAs for hypothesis 1, and two repeated-measures MANOVA for hypothesis 2.

ii. Aim 1 (Group Differences). Two omnibus MANOVAs revealed, contrary to our hypothesis, that groups were not significantly different in CT within the left hemisphere (Wilk's Lambda = .895, $F(4, 88) = 1.630$, $p = .174$) or within the right hemisphere (Wilk's Lambda = .878, $F(4, 88) = 1.474$, $p = .217$) . Therefore, there were not group differences between our autistic participants and their TD counterparts at the pre-time point for any of the of structures.

iii. Aim 2. An omnibus repeated-measures MANOVA for the left hemisphere structures indicated that Box's test of equality of covariance matrices was violated (Box's M = 39.064, $F(72, 2551.423) = 1.643$, $p = .036$), therefore Pillai's Trace was used. Results showed no significant main effect of time at the multivariate level (Pillai's Trace = .011, $F(2, 44) = .248$, $p = .782$). A significant time x group interaction effect was indicated at the multivariate level (Pillai's Trace = .190, $F(4, 88) = 2.367$, $p = .045$). Follow-up univariate tests showed the interaction was driven by the lOFC ($F(2,44) = 3.680$, $p = .033$, Partial $\eta^2 = .143$) as the mOFC was shown to not have a significant interaction ($F(2,44) = 2.137$, $p = .130$, Partial $\eta^2 = .089$). Paired sample t-tests, however, showed no significant change from pre- to post-test CT in the EXP ($t(19) = -1.670$, $p =$

.111), WL ($t(17) = 2.086$, $p = .052$, Cohen's $d = 1.165$), and TD ($t(8) = 0.803$, $p = .445$). An omnibus mixed methods repeated-measures MANOVA for the right hemisphere showed no significant main effect of time at the multivariate (Wilk's Lambda = .955, $F(2, 44) = 1.029$, $p = .366$), as well as no significant time x group interaction effect (Wilk's Lambda = .918, $F(4, 88) = .957$, $p = .435$).

Therefore, the hypothesis that the EXP group would show a change in CT from pre-test to post-test was not supported by the results. Refer to table 21 for means and standard deviations.

Table 21

Means and Standard Deviations for Group by Time Interaction of OFC Cortical thickness (mm) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre M(SD)	Post M(SD)	Pre M(SD)	Post M(SD)	Pre M(SD)	Post M(SD)	
Left							.061
Lateral OFC	2.67 (0.18)	2.76 (0.14)	2.81 (0.19)	2.74 (0.13)	2.78 (0.06)	2.76 (0.07)	.033
Medial OFC	2.62 (0.14)	2.68 (0.19)	2.66 (0.16)	2.65 (0.11)	2.66 (0.19)	2.55 (0.25)	.130
Right							.323
Lateral OFC	2.55 (0.29)	2.66 (0.11)	2.71 (0.20)	2.67 (0.15)	2.67 (0.15)	2.71 (0.14)	.125
Medial OFC	2.50 (0.19)	2.59 (0.17)	2.63 (0.16)	2.64 (0.13)	2.57 (0.16)	2.59 (0.17)	.343

D. Fusiform Gyrus (FFG sulci and gyri, and lateral FFG)

i. Screening. Eleven unique outliers (1.46% of data) were identified, two in the left FFG, four in the right FFG, four in the left lateral FFG, and one in the right lateral FFG. Two of the eight outliers were extreme outliers, both in the right FFG. One was Winsorized to the second lowest value; one was Winsorized to the next highest value (Tabachnick & Fidell, 2013). Subsequent analyses showed no significant difference in results regarding if the remaining mild outliers were trimmed, or not transformed. Therefore, the results presented are presented with outliers included.

ii. Aim 1 (Group Differences). A MANOVA at pre-test indicated that Box's test of equality of covariance matrices was violated (Box's M = 50.039, $F(20, 2574.123) = 2.099$, $p = .003$), therefore Pillai's Trace is utilized in subsequent results. Results showed no significant differences between groups (i.e., EXP versus WL versus TD) across the four structures (Pillai's Trace = .143, $F(8, 84) = .811$, $p = .595$). Therefore, the hypothesis that ASD groups would be significantly different in measures of structure thickness compared to their TD counterparts was not supported.

iii. Aim 2. An omnibus mixed methods repeated-measures MANOVA indicated that Box's test of equality of covariance matrices was violated (Box's M = 170.704, $F(72, 2103.419) = 1.581$, $p = .002$), therefore Pillai's Trace is utilized in subsequent results. No significant main effect of time at the multivariate level, (Pillai's Trace = .051, $F(4, 41) = .811$, $p = .701$), and no significant interaction effect (Pillai's Trace = .200, $F(8, 84) = 1.164$, $p = .331$) were found. Therefore, the hypothesis that structure CT would change over time was not supported. Refer to table 22 for means and standard deviations.

Table 22

Means and Standard Deviations for Group by Time Interaction of Fusiform Gyri cortical thickness (mm) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Fusiform Gyrus (FFG)							.331
Right FFG	2.86 (0.11)	2.91 (0.10)	2.90 (0.13)	2.86 (0.14)	2.92 (0.18)	2.91 (0.16)	.028
Left FFG	2.85 (0.16)	2.90 (0.13)	2.89 (0.09)	2.92 (0.12)	2.87 (0.11)	2.87 (0.10)	.666
Right Lateral FFG	2.88 (0.22)	3.00 (0.17)	2.98 (0.17)	2.95 (0.18)	3.04 (0.28)	3.00 (0.22)	.044
Left Lateral FFG	2.85 (0.22)	2.90 (0.16)	2.89 (0.19)	2.92 (0.17)	2.87 (0.19)	2.87 (0.17)	.533

E. Insula

i. Screening. Eleven outliers (5.85%) were identified in insula volumes, six in the left Insula, and five in the right Insula. Subsequent analyses showed no difference in results regardless if the remaining mild outlier was trimmed, or not transformed.

ii. Aim 1 (Group Differences). A MANOVA with both left and right Insula as dependent variables at pre-test revealed no significant differences between groups at the multivariate (Pillai's Trace = .086, $F(4, 90) = 1.008$, $p = .408$). Our hypothesis, that insula volumes between Autistic participants (i.e., EXP and WL) versus TD participants at pre-test would be significantly different, was not supported.

iii. Aim 2. An omnibus mixed model repeated-measures MANOVA showed no significant main effect of time at the multivariate level (Pillai's Trace = .004, $F(2, 44) = .086$, $p = .917$), and no significant interaction effect (Pillai's Trace = .084, $F(4, 90) = .968$, $p = .429$). Therefore, the hypothesis that the EXP group would show a change in thickness from pre-test to post-test was not supported by the results. Refer to table 23 for means and standard deviations.

Table 23

Means and Standard Deviations for Group by Time Interaction of Insula cortical thickness (mm) for all groups at Pre- and Post-Test

	Group (N = 47)						
	Experimental (n = 20)		Waitlist Control (n = 18)		Typically Developing (n = 9)		p
	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	Pre $M(SD)$	Post $M(SD)$	
Insula							.429
Left Insula	3.27 (.21)	3.31 (.14)	3.34 (.18)	3.33 (.15)	3.29 (.07)	3.29 (.10)	.474
Right Insula	3.20 (.25)	3.27 (.17)	3.33 (.12)	3.30 (.15)	3.28 (.12)	3.27 (.12)	.151

X. Structure Cortical Thickness and Behavioral Measures (Aim 3)

As no significant changes in CT were found for any of the structures, aim 3 was not applicable to these structures.

XI. Discussion

The current study sought to examine structural social brain differences, in a group of Autistic adolescents versus TD adolescents prior to the PEERS® intervention; the change in volume following a RCT of the PEERS® intervention; and associations between volumetric change over time and behavioral measures. Our first hypothesis, that structural differences would be present at pre-test between the ASD groups versus the TD group, with the exception of the volume of the amygdalae, was partly supported.

Amygdala volumes did not show any differences between groups, as hypothesized; however, our hypothesis t2hat there would be differences in volume between groups at this pre-test stage was not supported by any of the remaining structures. Prior research outcomes reporting on the difference in amygdala volume between autistic adolescents and their TD counterparts has eluded consistency. Studies have shown autistic adolescents having enlarged volume (Groen et al., 2010; Howard et al., 2000; Mosconi et al., 2009; Munson et al., 2006), decreased volume (Aylward et al., 1999; Nacewicz et al., 2006; Pierce et al., 2001; van Rooij et al., 2018), and no difference in volume compared to TD adolescents (Haznedar et al., 2000; Nacewicz et al., 2006; Schumann et al., 2004). It has been posited that these inconsistencies may be due to confounding factors, in the form of comorbidity, particularly anxiety disorders (Herrington, Maddox, Kerns, et al., 2017; Nacewicz et al., 2006). Therefore, as we could

not control for these extraneous factors due to our sample size, we hypothesized that there would be no difference between groups at pre-test as a result of random sampling. Our findings, contrary to some previous research and supportive of other previous research (e.g., Hyde et al., 2010; Tu et al., 2016; Zielinski et al., 2014), showed no difference between groups on any of the structures. This may be due to confounding factors such as those posited to affect amygdala volume studies (Herrington, Maddox, Kerns, et al., 2017; Nacewicz et al., 2006). It may also be a product of the rapidly changing brains and bodies of adolescents going through puberty, particularly considering prior research showing the effects of puberty on the adolescent brain (Blakemore, 2008; Blakemore et al., 2010). For the purpose of our study, however, it may have inadvertently provided an extra level of control for the future direction of our main aim (i.e., aim 2) which examined changes over 14 weeks of intervention, or waitlist-control for autistic adolescents and their TD counterparts. In having no significant differences between groups at pre-test, the future examination of morphology measures of group differences at post-test is facilitated.

A prior study examined changes in functional activity in preschoolers with ASD before and after undergoing 16-weeks of the Pivotal Response Theory intervention (Ventola et al., 2015), and found that, at post-intervention, the participants showed functional activation in the posterior STS more similar to their TD peers than at pre-test. However, no known study, to our knowledge, has examined structural morphological brain changes over a social skills intervention for individuals with ASD. Our hypothesis that social brain structures would show change in the EXP group from pre-intervention to

post-intervention time-point was supported by our investigation into volume, specifically bilateral amygdala volumes, left cACC volume, and left STG volume.

In regards to the amygdala, the present study showed decreased bilateral amygdalae volumes in the EXP group over the course of the PEERS® intervention. Although previous research of amygdala volume associations in adolescents with ASD have shown that smaller volumes were linked to greater challenges in social communication and emotion recognition (Baribeau et al., 2019; Nacewicz et al., 2006), no significant links to social communication measures (i.e., SRS subscale and SIAS) were found in the present study. It may also be that the common comorbidity found between social anxiety and ASD contributed to this pattern. As social anxiety in young adults without ASD, as well as in children with ASD, have been linked to an increased amygdala volumes (Juranek et al., 2006; Machado-de-Sousa et al., 2014), and PEERS® has been shown to decrease social anxiety in adolescents (Schohl et al., 2014), the decrease in amygdala may be related to those changes. This is to say, decreases in amygdala volumes in the EXP group may be a product of decreasing social anxiety in the participants, and not a product of changes in social skills and communication. Another possible alternative explanation may be that depressive symptoms, which have also been shown to be ameliorated by undergoing the PEERS® intervention, may contribute to the decreased volume in amygdala volumes at post-intervention timepoint. This may be the case, in a similar mechanism by which social anxiety symptoms may contribute to amygdala decreases since depression has also been shown to be related to increased amygdala volumes in autistic children (Juranek et al., 2006). Future studies should seek

to investigate social anxiety and depressive symptom severity at pre-test and post-test as a possible covariate for amygdala changes following the PEERS® intervention.

However, an alternative explanation, central to the stress inherent in interventions, could be posited. It may be that PEERS®, by making adolescents confront their social challenges via exposure, therefore potentially causing added stress, may induce a trauma response in the brain. As a prior study has shown that amygdala volumes showed decreased size in veterans with post-traumatic stress disorder (PTSD) compared to those that did not have PTSD (Morey et al., 2012), it may be that adolescents that undergo PEERS® may have a similar physiological response to the additional stress of being taught to initiate with social partners.

The significant increase over time of the left cACC in all the groups also supported the hypothesis that structure volumes would change following the PEERS® intervention. All groups may have shown a significant increase over time as a maturation effect and product of adolescence being a period of rapid growth. Nonetheless, the EXP group showed a greater change over time, as well as having a larger effect size, than the WL group, which may indicate that PEERS® contributes to the increasing size of the cACC above that found from maturation effects. Recent studies have shown a decrease in ACC (including the cACC) grey matter volume in adolescents and young adults with ASD compared to TD counterparts (Carlisi et al., 2017; Pereira et al., 2018). Furthermore, although no studies, to our knowledge, exist on relations between cACC volumes, and social communication, there have been functional activation and connectivity studies exploring these links (e.g., Pereira et al., 2018; Zhou et al., 2016). One of these studies showed a negative link between functional activation and

connectivity between the left cACC, specifically, to other social brain areas (e.g., STG and Insula), and social challenges (i.e., higher connectivity is associated with lower social challenges; Pereira et al., 2018; Zhou et al., 2016). Therefore, although our changes in volume were not associated with our measures of social challenges (i.e., SRS and SIAS), the increase in cACC volume provides support for further investigation of the moderation of social behavior changes by brain structure changes following PEERS®. More specificially, the increase in volume indicates a change of the cACC to approach more similar TD cACC volumes, instead of a stagnation or decrease in volume that may lead to the decreased grey matter volumes in ASD found by recent studies (Carlisi et al., 2017; Pereira et al., 2018). Furthermore, following the historically commonly used structural volume analogue for absolute neuron count, thus greater processing capabilities (i.e., greater functional activation, in neuroanatomy; e.g., Herculano-Houzel, 2009, 2012; Im et al., 2008; Roth & Dicke, 2005), an increased volume in the cACC may indicate an increased activation and functional connectivity. Future studies should investigate the change in cACC functional connectivity and activation over the course of the PEERS® intervention, and its relation to social challenges or the amelioration thereof.

Lastly, a significant increase over time in left STG volumes for the WL and TD groups, but not the EXP group, was found. Furthermore, we did not find any significant links between STG change over time and behavioral measures of social communication challenges (i.e., SRS and SIAS) or comorbid symptoms (CBCL subscales). However, given the current literature on STG grey matter volume and social communication links, the EXP group may be benefitting from not undergoing an increase over time, such as seen in the WL group. A prior study examining social brain structure volumes and their

relations to social behavior found, in autistic adults, a significant positive relation between Left STG and ADI-R Social and Communication Scores (Rojas et al., 2006). Therefore, it may be beneficial for adolescents to not show an increase in left STG volumes as they progress through the lifespan. As the WL and TD group both showed an increase in grey matter volume over time, whereas the EXP group did not, it may be that PEERS® contributes to behavioral changes that affect the neuroplasticity of the STG brain structure in a manner that limits or slows its growth. Furthermore, although we did not see behavior and STG volume links in this study, it may be, in part, due to the difference in the present measures (i.e., SRS, and SIAS) as opposed to the ADI-R used by Rojas and colleagues (2006).

No significant group x time interactions were observed in either SA or CT measures between groups across time points. Our most robust finding in the volume analysis (i.e., the amygdala) was not processed in a manner that SA or CT could be measured, the former a result of brain segmentation limitations of our atlas, and the latter a result of the amygdala being a subcortical structure. It is possible that a measure of SA of the amygdala may have provided greater detail into the morphological changes to this structure, and should be included as a measure in future studies. In addressing the nonsignificant findings of change of CT, and SA in the cACC and STG, despite the significant change in volume over time, it may be that volume changes resulted from areas not included in the CT and SA measures (i.e., volume could have changed without affecting the outermost parts of the structures). Future studies may look at different morphological measures of changes such as cortical folding and white matter tract

changes over time, as both of these may help provide a more detailed picture of neuroplasticity as it is affected by a social skills intervention.

Our third aim, to examine questionnaire reports of communication challenges and behavioral change in structures showing a significant time x group interaction in aim two, did not show any significant associations. No known study has examined change in structures over a social skill intervention for adolescents in relation to behavioral changes. Those that have examined links at a comparative descriptive study level (i.e., one time point) have most often used the ADOS, ADI-R, RMETS, Vineland, and Autism Quotient (e.g., Juranek et al., 2006; Munson et al., 2006; Rojas et al., 2006; Sato et al., 2017; Schumann et al., 2011; von dem Hagen et al., 2011), with only four identified studies utilizing the SRS, three in relation to CT and one in relation to functional activity (Prigge et al., 2018; Scherf et al., 2010; Tu et al., 2016; Wallace et al., 2012). Therefore, as the only morphology measures we utilized in Aim 3 were the volume measures, and the behavioral measures utilized were different than those used in previous volume studies, it may be that the present measures did not capture the same constructs as, for example the ADOS, ADI-R, and RMETS. Furthermore, due to our small sample size, a mixed methods repeated-measures MANCOVA was utilized instead of the more commonly accepted path analysis, which allows for multiple outcome variables to be examined at the same time whilst conserving power, thus providing estimate of associations across, in this case, structure change and behavioral change (Meehl & Waller, 2002). Future studies should attempt to a recruit sufficient sample size to increase power of the study overall, as well as use more appropriate statistical analyses.

XII. Limitations and Future Directions

Several limitations and future directions are abovementioned, however, one additional major limitation of the present study was the inability to control for comorbid diagnoses and symptoms that have been previously linked to abnormal "social brain" structure morphology (Herrington, Maddox, Kerns, et al., 2017; Juranek et al., 2006; Machado-de-Sousa et al., 2014; Morey et al., 2012). Although this study did originally recruit an equal amount of ASD only, and ASD + Anxiety in the EXP and WL group, it was not possible to examine these potential confounds due to having several MRI scans that were not processable due to motion occurring during the scan over and above that for which Freesurfer could correct. Future studies should recruit a more homogeneous pool of ASD-only groups to examine this issue. Alternatively, future studies should recruit equal sample pools of ASD-only versus ASD + Anxiety (or depression) for which to compare outcome differences. Furthermore, studies utilizing the latter suggestion should take care to control for length of anxiety, and depression comorbidity, as well as number of episodes of depression, as these factors can differentially effect amygdala volumes (i.e., an acute episode has been linked to increases in amygdala volumes whilst repetitive or enduring episodes have been linked to decreases in amygdala volumes; McEwen, 2003; Nacewicz et al., 2006). Another limitation is that this study did not employ questionnaire or behavioral measures of factors related to the specific structures that showed change over time. Future studies may benefit from including these types of measures to better understand the links between morphological changes and behavioral changes. For example, a measure of recognition of facial expressions and gaze direction, such as eye tracking and a facial expression recognition task, could be employed to better

understand the behavioral changes underlying the morphological changes in the STG. In addition, a measure of efficiency in acquiring emotional stimuli-reinforcer associations may help further elucidate the effects of a change in the cACC. Finally, a measure of emotion recognition to better understand behavior changes linked to change in amygdala volume may also be beneficial in further studies. Therefore, future studies should be cognizant of the effects of comorbid conditions and control for these effects, as well as employ more structure function specific behavioral measures.

In conclusion, our findings demonstrate strong support for neuroplasticity outcomes for *PEERS® for Adolescents* by way of changes on a volumetric level for social brain structure, particularly the amygdalae, left cACC and left STG. Furthermore, our findings increase the support for efficacy of *PEERS® for Adolescents*, utilizing a previously unstudied outcome measure, signaling change in social brain structures volumes previously shown to be linked to social and communication challenges. Furthermore, utilizing this type of outcome measures in future studies may help elucidate the differential effects of different social skill interventions.

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